The Risk Factors and Clinical Spectrum of Acute Myocardial Infarction among Females patients

By

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Dedication

TO the memory of

MY MOTHER

Whose inspiration always motivated me.

Acknowledgement

I am immensely grateful to Dr. Mohammed Sirag Abbasher for his long standing patience and encouragement.

I would like to thanks my friends Dr. Amani and Dr. Sara for their assistance to produce this work.

I am particularly grateful to my brothers and sisters and last but not the least I am obliged to all the patients and the CCU staff for their great support.

Content

List of Figures

List of Tables

Abbreviations

AF	Atrial Fibrillation
AMI	Acute Myocardial Infarction
BP	Blood Pressure
CAD	Coronary Artery Disease
CCU	Coronary Care Unit
CHD	Coronary Heart Disease
CHF	Congestive Heart Failure
CK	Creatine Kinase
DM	Diabetes Mellitus
ECG	Electrocardiogram
HB	Hemoglobin
HDL	High Density Lipoprotein
HF	Heart Failure
HRT	Hormone Replacement Therapy
IHD	Ischemic Heart Disease
LDL	Low Density Lipoprotein
LV	Left Ventricle
LVH	Left Ventricle Hypertrophy
MI	Myocardial Infarction
PAI	Plasminogen Activator Inhibitor
VF	Ventricle Fibrillation
VT	Ventricular Tachycardia

Abstract

This study was carried out at Al Shaab Teaching Hospital during the period between October 2003- June 2004.

The objective of this study is to determine the clinical presentation, risk factors and the immediate complication of acute myocardical infarction among Sudanese females.

In the final analysis it was found that most of the patient's age range between 50-70 years. The risk factors were the following: Diabetes mellitus (46%), systemic hypertension (40%), positive family history of IHD (23%) and lastly smoking (9%).

Anterior MI was the commonest type (55%) then comes inferior MI (38%) followed by posterior MI (7%).

In addition, it was found that more than half of the patients had elevated cardiac enzymes (CK-MB).

ملخص الاطروحة

هذه الدراسة الوصفية اجريت بمستشفى الشعب التعليمى خلال الفترة مـا بـين اكتـوبر 2003 الى يونيو 2004 ولقد خصصت هذه الدراسة لمعرفة العوامـل المـسببة للذبحـة القلبية لدى المراة السودانية.

شملت الدراسة مائة امراة تتراوح اعمارهن ما بين 40-70 سنة.

ولقد تبين ان مرض السكرى عامل رئيسى بنسبة 46%, بينما ارتفلع ضغط الدم يـشكل نسبة 40%, ثم التاريخ الاسرى نسبة 13% واخيرا التدخين بنسبة 9%.

و من الجدير بالذكر انه 55% من الحالات كانت الذبحة فى الجزء الامامى مـن عـضلة القلب بينما 38% فى الجزء السفلى و 7% فى الجزء الخلفى.

ACUTE MYOCARDIAL INFARCTION

DEFINITIONS:

Acute myocardial infarction (MI) is a focus of necrosis resulting from an in adequate tissue perfusion, it is accompanied by consequent hypoxia, accumulation of deleterious metabolites, and signs and symptoms of myocardial cell death. Anoxia without decreased perfusion can cause M.I but does so only rarely.

MI refers also to the typical clinical syndrome that results from such ischaemia and that is manifested by signs and symptoms such as crushing chest pain, diaphoresis, malignant ventricular arrhythmia, heart failure, and shock. However, MI can also present as sudden cardiac death, or a typically clinically silent or subtle, also with new onset or accelerated angina, Atypical chest pain mimicking (indigestion), impaired cerebral perfusion with syncope, and signs simulating those of a cerebrovascular accident or altered mental states.

Coronary thrombosis is recognized as a potential cause as early as 1910, and was established unequivocally as the pathophysiologic cause by early angiographic study of afflicted patients. [(1)]

PATHOPHYSIOLOGY:

The most common cause of AMI is narrowing of the epicardial blood vessels due to atheromatous plaques rupture with subsequent exposure of the basement membrane results in platelet aggregation, thrombus formation, fibrin accumulation, haemorrhage in to the plaque, in addition to varying degrees of vasospasm. This can result in partial or complete occlusion of the vessel for more than 4-6 hours and results in irreversible myocardial necrosis, but reperfusion within this period can salvage the myocardium and reduce morbidity and mortality. [(2)]

FREQUENCY:

In the US, AMI is a leading cause of morbidity and mortality. Approximately 1.3 million cases of nonfatal AMI are reported each year, for an annual incidence of approximately 600 per 100,000 people.

MORTALITY/MORBIDITY:

- Approximately 500,000-700,000 deaths caused by ischemic heart disease occur in the United States alone.
- More than one half of deaths occur in the pre hospital setting.

- In-hospital, fatalities account for about 5-15% of all deaths. An additional 10% of deaths occur in the first year post infarct.

RISK FACTORS:

- Gender

  Male predilection exists in persons aged 40-70 yeas.

  In persons older than 70 years, no sex predilection exists.

- AGE:

  AMI occurs most frequently in persons older than 45 years.

Certain subpopulations younger than 45 years are at risk, particularly: cocaine users, insulin dependent diabetics, patients with hypercholesterolemia, and those with a positive family history of early coronary disease. A positive family history includes any first-degree male relative aged 45 years or younger or any first-degree female relative aged 55 years or younger who experienced a myocardial infarction.

In younger patients, the diagnosis may be hampered if the physician does not maintain a high index of suspicion.

HISTORY:

- Chest pain, usually pressuring, or squeezing.
- Pain may radiate to the jaw, neck, arms, back, and epigastrium. The left arm is affected more frequently than the right arm.
- Dyspnoea, which may accompany chest pain or occur as an isolated complaint, indicates poor ventricular compliance in the setting of acute ischemia
- Nausea and/or abdominal pain often are present in infarcts involving the inferior wall.
- Anxiety.
- Light headedness and syncope.
- Cough.
- Nausea and vomiting.
- Diaphoresis.
- Wheezing.
- Elderly patients and those with diabetes may have particularly subtle presentations and may complain of fatigue, syncope, or weakness.

PHYSICAL EXAMINATION:

- Frequently, physical examination findings are normal.

- Patients with ongoing symptoms usually will lie quietly in bed, appear pale and diaphoretic.
- Hypertension may precipitate AMI, or it may reflect elevated catecholamines due to anxiety, pain, and exogenous sympathomimetics.
- Hypotension indicates ventricular dysfunction due to ischemia, it usually indicates a large infarct and may be observed with a right ventricular infarct.
- Acute valvular dysfunction may be present ;
- This dysfunction usually involves the papillary muscle.
- Mitral regurgitation due to papillary muscle ischemia or necrosis may be present.
- Congestive heart failure {CHF} may occur;
- Neck vein distension.
- Third heart sound.
- Rales on pulmonary examination.
- New or worsening mitral regurgitant murmur may be noted.
- A fourth heart sound is a common finding in patients with poor ventricular compliance that is due to a pre-existing heart disease or hypertension.
- Dysrhythmias may be present;

With heart block and right ventricular failure, cannon jugular venous a waves may be noted.

CAUESE of MI:

- The predominant cause is a rupture of an atherosclerotic plaque with subsequent spasm and clot formation.
- Ventricular hypertrophy e.g. (left ventricular hypertrophy [LVH], idiopathic hypertrophic subaortic stenosis [IHSS], and an underlying valve disease).
- Hypoxia due to carbon monoxide poisoning or acute pulmonary disorders {infarcts in this setting usually occur when myocardial demands are dramatically increased relative to blood supply}.
- Emboli to coronary arteries, which may be due to cholesterol or infectious causes.
- Coronary artery vasospasm.
- Arteritis.
- Coronary anomalies, including aneurysms of the coronary arteries.
- Cocaine, amphetamines, and ephedrine.
- Increase after load or inotropic effects, which increase myocardial demand.
- Primary vasospasm of the coronary artery.
- Risk factors for atherosclerotic plaque formation can be divided in to :

- FIXED:

  Age.

  Being a male and younger than 70 years.

  Family history.

- ALTERABLE:

  Smoking (Cigerate, Pipe. Shisha)

  Poorly controlled hypertension.

  Hyperlipidaemia.

  Diabetes mellitus.

  Type A personality.

  Sedentary lifestyle.

  Central obesity.

Laboratory Studies:

*Creatine kinase –MB (CK-MB)

- This is the criterion standard for detection of myocardial necrosis.

- Levels begin to rise within 4 hours after injury, peak at 18-24 hours, and subside in days.

- Upper limit of reference range values for CK-MB is 3-6% of total CK.

- Serial sampling over periods of (6-9) hours will increase the sensitivity to nearly 90%.

- Over 24 hours, the sensitivity is nearly 100 %. [(2)]

EFFECT OF MI ON ORGANS OTHER THAN THE HEART:

Augmentation of pulmonary venous pressure may cause diminished pulmonary compliance, dyspnoea,pulmonary vascular redistribution {detectable radiographically}, interstitial or alveolar pulmonary odema, respiratory decompensation and hypoxemia. Cerebral hypoperfusion may result in restlessness, or rarely psychosis coupled with dyspnoea in the elderly, it may be manifested only as confusion and combativeness.

Increased sympathoadrenal tone reflected by markedly elevated plasma catecholamines levels and adrenocortical stimulation may be prominent as well.

Plasma concentration of atrial natriuretic peptide decreases initially but then it increase,perhaps because of heart failure and atrial stretch.

Elevated plasma concentrations of vasopressin ,angiotensin{with B adrenergic stimulation of rennin release},and aldosterone contribute to fluid retension and hyponatremia.

Impaired pancreatic blood flow inhibits insulin secretion.

In addition to the typical increase in erythrocyte sedimentation rate and leukocytosis, modestly increased levels of plasma fibrinogen and augmented circulating plasminogen activator inhibitor type 1{PAI}-1 occur as part of the acute phase reaction to MI. Impaired fibrinolysis and augmented platelet activation by circulating catecholamines may predispose to continuing coronary and ventricular mural thrombosis. Plasma viscosity increase is due to increase in fibrinogen, alpha 2 globulins, and haemo-concentration, several days after the onset of MI, most markedly when left ventricular failure or shock supervenes.

FEMALES AND HEART DISEASE:

In females coronary heart disease (CHD) is responsible for one in every five deaths in the states in 1997. Long thought of as primarily affecting men, but we now know that CHD also affect a substantial number of women. More women US die from heart disease than any

other cause. Experts estimate that 1 in 2 women will die of heart diseases or stroke compared with 1 in 25 women who will die of breast cancer.

Current statistics reveals significant differences between males and females in survival following a myocardial infarction, for example 42% of females who have myocardial infarction die within 1 year compared with 24% of males. The reasons for this are not well understood. The explanation accepted by many is that women tend to develop heart disease later in life than do men who are likely to have coexisting chronic conditions. However research also has shown that women may not be diagnosed or treated as aggressively as men, and their symptoms may be very different from those of men who are having a heart attack. In addition new studies indicate that men and women react to drugs prescribed for heart disease and other conditions quite different; drugs that may help men can have serious side effects in women.[3]

RISK OF MI:

The aetiology of coronary artery disease (CAD) is multifactor; a number of risk factors are known to predispose to the condition, some of which cannot be changed such as: Age, Gender, Race, and Family history. Whereas other major risk factors such as: serum cholesterol,

smoking, central obesity, diabetes, and hypertension can be modifiable. However not all patients with MI are identified by these risk factors. [4]

Risk Factor of myocardial infarction in females:

A / Age:

Acute myocardial infarction occurs most frequently in persons older than 45 years.

Certain sub-population younger than 45 years are at risk, such as those with hypercholesterolemia or with positive family history for early coronary diseases.

Positive family history include any 1st degree male relative aged 45 years or younger, also any 1st degree female relative aged 55 years or younger who experienced myocardial infarction. [1]

Males have higher incidence of coronary artery diseases than premenopausal females. However after the menopause, the incidence of atherosclerosis in females approaches that in males. The reason for this gender difference are not clearly understood but probably related to the loss of the protective effect of oestrogen.

Females have more favorable risk profile in some respect:

-higher HDL cholestrol level.

-lower triglyceride level.

-less upper body obesity than males.

But females also have less favorable risk profile in other respects:

-Obesity.

-Higher blood pressure.

-Higher plasma cholestrol levels.

-Higher fibrinogen levels.

-Higher incidence for diabetes.

The explanation for gender differentiation in IHD is the 'cardioprotective' effect of oestrogen which could be due to suitable lipid profile, direct vasodilatation effect or perhaps other factors.

HDL cholestrol levels appear to be particularly important risk factor in females, it is higher in all age groups of females especially premenopausal and estrogen-treated postmenopausal females.

Smoking is another important factor in females.

The incidence of IHD increases markedly at menopuase, consistent with the hypothesis that estrogens are cardioprotective. A number of observational studies have supported this hypothesis by demonstrating significant decreases in IHD in females on hormone replacement therapy (HRT) either estrogen only or in combination with progestron. However the HERS resent clinical trial of HRT for the secondary prevention of IHD, showed no significant difference in cardiovascular events between therapy with combined continuous conjugated eguine estrogen and that

with medroxy progesterone acetate , compared to placebo over four years. Indeed, in the HRT group there was about 50% increase in cardiovascular events in 1st year of trial.

The females Health Initiative is investigating directly the impact of various HRT modalities as a primary prevention of IHD risk until further data are available , cautions should be excersised in prescribing HRT to females with a history of IHD or cardiopretective alone. (5)

It appears clear that estrogen deprivation increase the risk of cardiovascular disease in females. Data from the Framingham study have been used to compare the incidence of cardiovascular disease in males and females. Although the incidence is three times lower in females than in males before the menopause, it is approximately equal in males and females aged 75 to 79 being 53 and 50.4 / 1000 per year, respectively. This trend also pertains to gender difference in mortality due to cardiovascular disease. Coronary artery disease is the leading cause of death in females and the lifetime risk of death is 31% in postmenopausal females versus 3% risk of breast cancer.

Although cardiovascular disease became more prevalent only in later years following natural menopause, premature cessation of ovarian function (before menopause) constitutes a significant risk. Premature menopause, occurring before age of 35, has been shown to increase the

risk of myocardial infarction two to threefold and premature oopherectomy (before age of 35) increase the risk sevenfold. [6]

Myocardial infarction has been recognized for long time as the leading cause of death among middle aged males but only recently it has been recognized as an equally important cause of death and disability among older females. By age of 60 years, only 1 in 17 females in the United States had a coronary event, compared with 1 in 5 males. However after age of 60, coronary diseases become the leading cause of death among females. In this age group 1 in 4 males will die as result of myocardial infarction. [7]

In a study done in Hvidore, Denmark in 1996, they found that females with acute myocardial infarction tend to be older than males. They are on average 4 years older than males with acute myocardial infarction. Some investigation suggested that the protecting role of premenopausal estrogen against ischemic heart disease explain the later manifestation of myocardial infarction in females, whereas others regard differences in life style, including smoking habits, to play an important role. [8]

Heart attack mortality is higher for females than males before age of 60, but opposite is true for females and males older than age of 79. Age was a significant predictor of death even after adjustment for numerous dimorphic and clinical characteristic of pattern and the

treatment they received. Non-biological factors may play a role, including behavioral, psychological, social factors such as smoking, adherence to medication regimens, depression, social isolation low income and emotional stress.

It has been reported in the literature that the isolated value of LDL–cholesterol plays a less significant role as a cardiovascular risk factor in females as compared with that in males. On the other hand, low LDL-cholestrol level has been considered as an important predictor of mortality among females. Currently a meta-analysis carried out at the National Heart, Lung and Blood Institute has shown that total hypercholesterolemia and high LDL-cholestrol levels correlated with a higher cardiovascular mortality in females younger than 63 years old, but not in the elderly. [9]

In a study done in 20th of July 1996, in Denmark, among 700 women and 2300 men, it showed that females experiencing acute myocardial infarction tend to be older than males and that early mortality, 10 years mortality, re-infarction rate, mortality after re-infarction or cause of death do not differ between sexes. In conclusion, this study indicates that sex itself is not an indicator of the risk after acute myocardial infarction. [8]

B / Diabetes Mellitus:-

Diabetes is a risk factor in the development of myocardial infarction, atherosclerosis, stroke and amputation. The risk is related to that of the background population. For examples, Japanese diabetics are much less likely to develop atherosclerosis than patients in Europe, but are much more likely to develop it than non-diabetic Japanese.

Myocardial infarction is 3-5 times as likely and females with diabetes lose their pre-menopausal protection from coronary artery disease. (4)

Estrogen enhance insulin sensitivity in females but not in men. Despite this, prevalence of type 2 diabetes mellitus (DM) is higher in females who are related in part to the higher prevalence of female obesity. Pre-menopausal females with DM lose the cardioprotective effect of their female gender and have identical rates of IHD to those in males. This is partially explained by the presence of several IHD risk factors in females with DM = obesity, hypertension and dyslipidemia – recent evidence suggests that vascular responses differ in females with DM, as compared to normal females. (6)

DM and hypertension are less common in young patients. However, young patients commonly have subtle problems with glucose metabolism.

In one study of 108 patients without history of diabetes mellitus who had an M.I. before the age of 45. 65 percent had decreased oral glucose tolerance and a hyper-insulinemic response to oral glucose challenge. [10]

Cardiovascular complications e.g. (myocardial infarction) are the leading cause of morbidity and mortality related to DM. complications of atherosclerosis are responsible for about 80% of death in diabetic patients and 75% of hospitalizations. In a 7 years study conducted in Finland [11], the absolute risk for major cardiac events in patients who had type 2 diabetes without coronary artery disease (CAD) was 20.2% compared with 18.8% in matched non-diabetic patients who had established CAD, even before the development of hyperglycemia / persons with impaired glucose tolerance have an elevated risk of macro-vascular disease. Fifty percent of patients have evidence of cardiovascular disease at the time of diagnosis of type 2 diabetes. [12]

A study published in the Brazil (2002) indicates that DM was least prevalence risk factors (in young males and females with acute myocardial infarction) Present in 10% of the patients of both sexes. One is possible justification for the lower prevalence of diabetes mellitus in young patients of both sexes after acute myocardial infarctions could be the fact that the atherogenic effects of DM on the cardiovascular system appears through the years. [13]

Good evidence suggests that hyperlipidemic, diabetes and hyperhomocystinemia are risk factors for CHD in females. Risk factors for CHD seen to be equally strong in males and females with the possible exception of the age, diabetes, and certain lipoproteins.

C / Hypertension:

Patients with hypertension die prematurely; the most common cause of death is heart disease, coma with stroke and renal failure. Angina pectoris may also occur because of the combination of accelerated coronary arterial disease and increase myocardial oxygen requirement as a consequence of the increased myocardial mass.

Evidence of Ischemia or infarction may be observed late in the disease.

Most heart deaths due to hypertension results from myocardial infarction or congestive heart failure. Recent data suggest that some of the myocardial damage may be mediated by aldosteron in the presence of a normal / high salt intake rather than just the increased blood pressure or an increase in angiotension II level per se. [(6)]

Effective treatment of hypertension significantly reduces cardiovascular mortality and morbidity. The relative risk of stroke is

reduced by up to 40% and M.I by 20%. Although the incidence of M.I exceeds that of stroke the absolute risk reductions are similar. [14]

Hypertension is more common in U.S. females than males, largely owing to the high prevalence of hypertension in older age groups and longer survival rate for women. Both the effectiveness and adverse effects of various antihypertensive drugs appear to be comparable in females and males. Benefits of treatment for severe hypertension have been dramatic in both females and males. However, in clinical trials of the treatment of mild to moderate hypertension, females has had smaller decrease in morbidity and mortality than males, perhaps because females have a low risk of M.I and stroke than males, but it was found that older females benefit at least as much as males from treatment, which was demonstrated by the systolic hypertension in elderly study. The incidence of hypertension (above 140/90) appears to be low (less than 5%) with the current low-dose & oral contraceptives. Postmenopausal estrogen therapy is not associated with increase in the blood pressure. [6]

In another study, done by CoronadoBe, Griffith II (1997) of nearly 11000 older patients (half of which were females) who arrived with cardiac symptoms. It was found that females had more hypertension (55 percent vs. 46 percent) and diabetes (23 percent vs. 17 percent) compare to men but, had suffered fewer previous heart attacks (21 percent V-S -29 percent). They also had more heart damage than males. [15]

In the elderly program of systolic hypertension, in which females made up to 57% of the study population, antihypertensive treatment resulted in a 36% reduction in the incidence of stroke and 25% reduction in that of CAD. [7]

Cigarette Smoking & Health:

Cigarette smoking acts synergistically with other cardiac risk factors to increase the risk of Ischemic heart disease. Although the risk of cardiovascular diseases is roughly proportional to cigarette consumption, the risk persist even at low levels of smoking, that is, one to two cigarette per day. Cigarette smoking reduces exercise tolerance in patients with angina pectoris and intermittent claudication. Vasospastic angina is more common and the response to vasodilator medication is impaired in patients who smoke. The number of ischemic episodes and total durations as assessed by ambulatory electrocardiographic monitoring in patients with coronary heart disease are substantially increased by cigarette smoking. The increase in the relative risk of coronary heart disease because of cigarette smoking is greatest in young adults, who in the absence of cigarette smoking would have a relatively low risk. Females who use oral contraceptives and smoke have a synergistically increased risk of both myocardial infarction and stroke.

After acute M.I, the risk of recurrent myocardial infarction is higher and survival is half over the next 12 years in persistent smokers as compared with quitters. Smoking interferes with revascularization therapy for acute M.I. after thrombolysis, and the reocclusion rate is four-fold higher in smokers who continue than in those who quit. The risk of reocclusion of coronary artery (after angioplasty or a bypass graft) is increased in smokers. Cigarette smoking is not a risk factor for hypertension per se but does increase the risk of complications, including the development of nephrosclerosis and progression to the malignant hypertension. [16]

Studies of smoking and cardiovascular disease may have underestimated the risk in females because until recently females smoking habits have differed greatly from males. However, during the past few decades, females have taken up smoking like males. Using data from three large prospective studies in Copenhagen that included many females who were heavy smokers, (Prescott et al) found that the smoking related risk of myocardial infarction was 50% higher in females than in males. This difference may be caused by interactions between

In a study published in England, Scotland and Wales (1999), about risk of M.I in young female smokers, there was no interaction of smoking with the use of oral contraceptives, but there were additive risks with other clinical risk factors such as hypertension and diabetes. It is

estimated that if all females aged 16-44 years were able to stop smoking 1400 cases of M.I per annum (of whom 112 would died) would be prevented. In conclusions, in young females the risk of M.I from smoking was considerable and heavy smokers with other risk factors were especially at risk. [17]

In a study done by Stone, Shapiro [18] to examine the relation between M.I and cigarette smoking in young females, they investigated the smoking habits of females under age of 50 who had survived a recent M.I. they had not bear using oral contraceptives, and other identifiable risk factors were excluded. Among 55 such females and 220 controls matched for age and area of residence, the proportions of cigarette smokers were 89 percent and 55 percent respectively (P less than 0.001).

A Dose response relation was evident, among females smoking 35 or more cigarette per day the rate of M.I was estimated to be some 20-fold higher than among those who had never smoked. This study demonstrated that cigarette smoking is a risk factor for M.I in young females who are otherwise apparently healthy.

Other study established in Denmark, Copenhagen City heart study[19] to compare risk of M.I associated with smoking in males and females taking into consideration differences in smoking behavior and a number of potential confounding variables, they found that females may be more sensitive than men to some of the harmful effects of smoking.

Interactions between components of smoke and hormonal factors that may be involved in the development of Ischemic heart disease should be examined further, which was found as a possible cause of the sex differences.

There is a growing epidemiological evidence that females who smoke are relatively deficient in estrogen, they have an earlier menopause, decreased risk of cancer of the endometrium, greater likehood of osteoporosis (osteoporotic fractures), and reduced incidence of a number of minor" disorders such as uterine fibroids.

Recently found that the relative mortality from vascular diseases was higher in a female smoker than in a male smoker. [20]

In a study done by Eva Prescott, Merete Hippe (April 1998), they found that sex difference in the effect of tobacco can not simply be put down to differing base line rates. Although relative risk are higher in females, suggesting differences in mechanism of action of tobacco in males compare to females, smoking may well cause more cases of M.I among males in this study population. The difference in risk was higher in males to age of 65 and in females after age of 65. [21]

Other Factors:

A variety of other possible contributing factors have been identified in young patients with M.I these include:

Oral contraceptive: use in young females, particularly in combination with smoking. [22]

Defective fibrinolytic function: mainly due to elevated plasma plasminogen activator inhibitor -1 ((PA/ -J)) activity. [23]

The role of psychosocial and other social factors in M.I:

Life events and other crisis: one report in 1975 found that stressful life events occurred among 40 of 100 sudden dearth victims in 24 hours preceding death. [24] Since this and related studies are subjects to the criticism of biased recall by relatives or friends of the victims. prospective studies are somewhat more reliable.

In a large cohort of middle- aged widowers, for example, a 40 percent increase in the mortality rate in the first 6 months was observed following bereavement, more than half of which was attributed to cardiovascular causes. [25] It is unclear how much of this increase in mortality was due to lifestyle changes during grieving period rather than to stress per se.

Personality and mood: studies of the role of personality in the etiology of coronary artery disease had yielded controversial results. However, there is increasing evidence that hostility, cynicism and anger

from a critical “toxic” component of type A behavior is associated with enhanced cardiovascular risk. [26]

Depression:

Although there is mounting evidence that depressive symptoms are associated with increased cardiovascular risk, it remains uncertain whether the symptoms are playing a causative role or whether they are primarily a marker or a prodrome of an evolving event. As early as 1937, the mortality among 1900 patients hospitalized for depression was found to be six fold than that of an age- Matched population. More recent studies have confirmed this association. [27]

Seasonal Pattern:

Several series have demonstrated a seasonal pattern of death from M.I, with more fatal events (20 to 30 percent variation) occurring in the winter than summer. As an example, a recent report from the National Registry of myocardial infarction evaluated 259, 891 cases from 1474 hospitals during a 25 months period, approximately 53% , more infarctions occurred in the winter or spring compared to the summer. The trend were independent of gender, geographic location, age, and the type ((Q wave or non- Q wave)) of M.I. In hospital, fatality rates from M.I

also followed a seasonal pattern, with a peak of 9 percent in winter and a nadir in the spring.

However, a seasonal pattern is absent in diabetics or those taking beta blockers or aspirin, suggesting an important role for the sympathetic nerves system. [28]

Risk stratification for cardiac events after acute myocardial infarction:

The process of risk stratification in patients who has had an acute M.I has two components:

- Identification of patients at increased risk for recurrent Ischemic events.
- Identification of patients at increased risk for arrhythmic or non-arrhythmic death. Arrhythmias, for example, are the most frequent cause of death during the first year after an M.I.

Early risk stratification: risk stratification of the post- M.I patient begins as soon as the patient arrives to the coronary care unit (CCU). There are many clinical prognosis factors that are immediately available to the physician based upon the initial history, physical examination,

electrocardiogram (ECG) and chest x-ray. Most of these prognostic factors have been recognized for over 30 years.

- Heart rate: Although heart rate may be elevated soon after the patient arrives in the C.C.U, it usually declines thereafter to a level which reflects the degree of activation of sympathetic nervous system.

Patients with sustained heart rates >90 beats/min usually have marked impairment of left ventricular (LV) function and therefore, a poor prognosis. [29]

- Heart failure: evidence of heart failure (HF) on physical examination or chest x-ray indicates extensive LV systolic and /or diastolic dysfunction and is associated with worse prognosis compared to those with no pulmonary congestion. Right ventricular HF or biventricular HF is associated with an elevation in jugular venous pressure.

Killip class: the killip classification, published in 1967, categorizes patients with an acute M.I based upon the presence or absence of simple physical examination findings that suggest LV dysfunction[30]:

Class I – no evidence of HF

Class II – findings consistent with mild to moderate HF (S_3, lung rates less than one-half way up the posterior lung fields, or jugular venous distension)

Class III - overt pulmonary edema

Class IV - cardiogenic shock.

The higher the killip class, the greater the in-hospital mortality. The killip class remains prognostically important, even in patients who undergo thrombolytic or primary percutaneous coronary intervention (Angioplasty with or without stenting).

- Hypotension :

Hypotension (systolic pressure < 100 mm Hg) or frank shock was associated with a poor prognosis. This is especially true in the patient with an anterior M.I in whom hypotension is the result of extensive myocardial damage and markedly reduced LV ejection fraction (LVEF). In comparison, hypotension in the presence of an inferior M.I may be the result of increased vagal activity, the so-called Bezold-darisch reflex.

- New Q waves on the ECG:

In patients with an ST elevation acute M.I., the presence of pathologic Q waves on the ECG signifies an irreversible myocardial necrosis. However, the prognostic importance of new pathological Q waves on the presenting ECG is uncertain.

This was examined in one study of 481 patients presenting within four hours of symptoms onset. New Q waves were associated with lower

LVEF, an increased 30 days cardiac mortality, and an increased mortality after a five years follow –up (31).

Arrhythmia:

The presence of certain arrhythmias beyond the first 24 to 48 hours post-MI predict a worse prognosis. These include a trial fibrillation (AF), ventricular tachycardia (VT), and ventricular fibrillation (VF).

AF is the most common supra ventricular arrhythmias after an M.I. The GISSI- Z trial, for example, evaluated over 11,000 patients treated with thrombolytic therapy for acute M.I. [32]

TIMI risk score:

The TIMI risk score, based upon data from 15,000 patients with an ST segment elevation M.I eligible for fibrinolytic therapy, is a simple arithmetic sum of eight independent predictors of mortality. [33]

Stress hyperglycemia:

There is a positive association between hyperglycemia at the time of an MI and mortality. The evidence that the use of insulin to lower glucose concentration reduces mortality in patients with diabetes suggests that hyperglycemia is not only an epiphenomenon of stress response mediated by sympathetic stimulation. [34]

Prognosis after acute myocardial infarction:

It is well established that females with M.I have a worse prognosis than men, but disagreement persists about whether this reflects differences in age, risk profiles, or treatment given to females with M.I are typically older than men, and some investigators have found the differences in age, sufficient to account for all their excess risk. Also, hypertension and diabetes may be more common in females, and this factor too may contribute to the prognostic imbalance between the sexes. More recently, evidence has shown that females with coronary heart disease tend to be treated less intensively than males. If this applies to use of thrombolysis and drugs such as aspirin and B blockers, it may not only help explain females less favorable out come but also provide a relatively simple remedy for correcting it.

Although females with M.I have a worse prognosis than males, it is not known for how long after the acute phase the excess risk persists. Certainly, the survival curves for females and males presented by previous investigators are in most cases approximately parallel after initially diverging, which implied that the excess risk in females is a temporary phenomenon that eventually disappears. It is important, however, that the time it takes to disappear is defined and it's specific

strategies are to be developed to protect females with acute M.I during the period of heightened risk.

Sex related difference in short and long term prognosis after acute M.I:

In a study done by Galatius – Jensen, Registrare, JLaunbjerg, senior registrare [35], they found that: sex by itself is not a risk factor after acute M.I. females and males have similar mortality at 10 years follow up. Causes of death are not different between both sexes. Females and males have similar re-infarction rates and similar subsequence mortality. The prevailing view regarding sex as an independent prognostic factor after acute M.I may be due to present difference in treatment of females and males, selection bias, and the interpretation of the role of age differences.

In other study done by Ayaniant JZ (2001) [36], they found that heart attack mortality is higher in females than males before the age of 60, but the opposite is true for females and males older than age 79. Age was a significant predictor of death even after adjustment for numerous demographic, clinical characteristics of patients and the treatments they received. Non biological factors may play a role, including behavioral, psychological, and social factors such as smoking, adherence to

medication regimens, depression, social isolation, low income, and emotional stress.

Females are 20 percent more likely than males to die in the hospital following a heart attack. [37] This study of overt 12,000 females and males treated for heart attack in Seattle area hospitals between 1988 and 1994 found that even after accounting for differences between males and females in cardiac procedures, age and health factors, females were still 20 percent more likely to die than males. They also were less likely than males to receive clot-busting (thrombolytic) therapy promptly or bypass surgery.

Females received less aggressive treatment than males after a heart attack and are more likely to die while in hospital. [38] This study found that females continue to lag behind males in both treatment received and outcomes. Differences in age and severity of illness did not explain the variations seen in this study, which involved more than 14,000 patients admitted for heart attacks to 100 U.S hospitals in 1991.

Lack of studies on females limits usefulness of research on coronary heart disease:

Press release date: July 10/2003.

Although coronary heart disease (CHD) is the cause of more than 250,000 deaths in females each year, much of the research in the last 20 years on the diagnosis and treatment of CHD has either excluded females

entirely or included only limited numbers of females and minorities. As a result, many of tests and therapies that are used to treat females for CHD are based on studies conducted predominantly in males.

Objectives

- To identify the main clinical presentation of myocardial infarction among the Sudanese female.

- To determine the risk factor of acute myocardial infarction among the Sudanese females including family history, diabetes mellitus, hypertension and smoking.

Methodology

Study area: Al Shaab Teaching Hospital.

Study period: October 2003- June 2004.

Population:

All females' patients admitted to coronary care unit (CCU) with acute myocardial infarction during this period.

All female patients admitted to CCU were interviewed, examined and investigated.

Data collected using questionnaire.

The questionnaire included personal data and the risk factors of MI (diabetes mellitus, hypertension, hyperlipidemia, smoking and positive family history).

ECG was done for each patient.

Blood sample was taken for blood film of malaria, blood glucose, cardiac enzymes and lipid profile.

Inclusion criteria:

All females presented with acute transmural myocardial infarction, as evidenced by ECG changes (Q formation and ST segment elevation).

Exclusion criteria:

Females with other cardiac diseases (congenital and valvular heart disease and myopathies) and ECG changes of Bundle Branch Block were excluded.

Sampling:

Randomized 100 cases of female patients with acute MI were selected.

Data collection and analysis:

The data was collected by a self administered simple questionnaire and then analyzed and represented in a statistical manner.

RESULTS

This study was conducted on 100 female patients with myocardial infarction. 21% of the patient between 40-50 years, 35% between 51-60

years, 33% between 61-70 years and 11% more than 70% years **(Figure 1).**

Most of the patients 59% originally from Northern Sudan, 26% from Central, 21% from Western, 2% from Southern and 1% from Khartoum **(Figure 2)**.

89% of the patient were housewife, while 5% employed, 55% labourer and 1% others **(Figure3).**

The patient were married is 49%, widow 39%, divorced 8% and 4% were single **(Figure 4)**.

The patient were illiterate 62%, 22% had a primary school education level, 13%, intermediate school and only 3% completed the university studies **(Figure5).**

The distribution of the patients according to their number of pregnancies were 20% 1-3, 43% 4-6 and 37% > 6 **(Figure6)**.

Distribution of presenting symptoms, 92% presented with chest pain, 32% presented with shortness of breath and 24% presented with palpitation **(Figure 7, 8, 9).**

The patients that had a family history of ischemic heart disease is only 28% **(Figure 10)**.

The risk factors of smoking are shown in **(Figure 11, 12**), 9% of the patients used to smoke and duration more than 10 years in 55% of patients.

(Table 3) showed that who used to smoke were 11% in the age group 40-50, 44% in 51-60 years and 12% more than 70% years.

The distribution of systemic hypertension as a risk factor is shown in **(Figure 13-14)**, 40% had systemic hypertension and duration more than 15 years in 23%.

(Table 4) showed that those were known to be hypertensive were 19% in age group 40-50 years, 35% in 61-70 years and 11% more than 70 years.

Diabetes mellitus occurred in 46% with a duration of more than 15 years in 35% **(figure 15 and table 1)**, 10% of them are in age group more than 70% years **(table 2)**.

Distribution of presenting signs, 34% presented with high blood pressure, 4% had a sign of hyperlipdaemia and 16% of the patient were pale on admission **(Figure 16-17)**.

(Table 6) showed that 12% of the patients in the age group 40—50 presented with pallor, 19% in 51-60 years, 50% in 60-70 years and 19% more than 70 years.

Only 3% of the patient presented with arrthymias and 6% presented with sign of heart failure **(table 7 and 8**).

Random blood sugar was high in 44% **(figure 19).** Normal Hb% in 80% and 20% had low Hb% **(figure 20).** BFFM positive in 12% of the patients while 88% had negative result **(Figure 21%).**

ECG showed 55% of the patients had anterior MI, 38% had inferior M.I, while 7% had posterior M.I **(table 9).**

Cardiac enzyme are high in 42%, while 58% had abnormal result **(table 10)**.

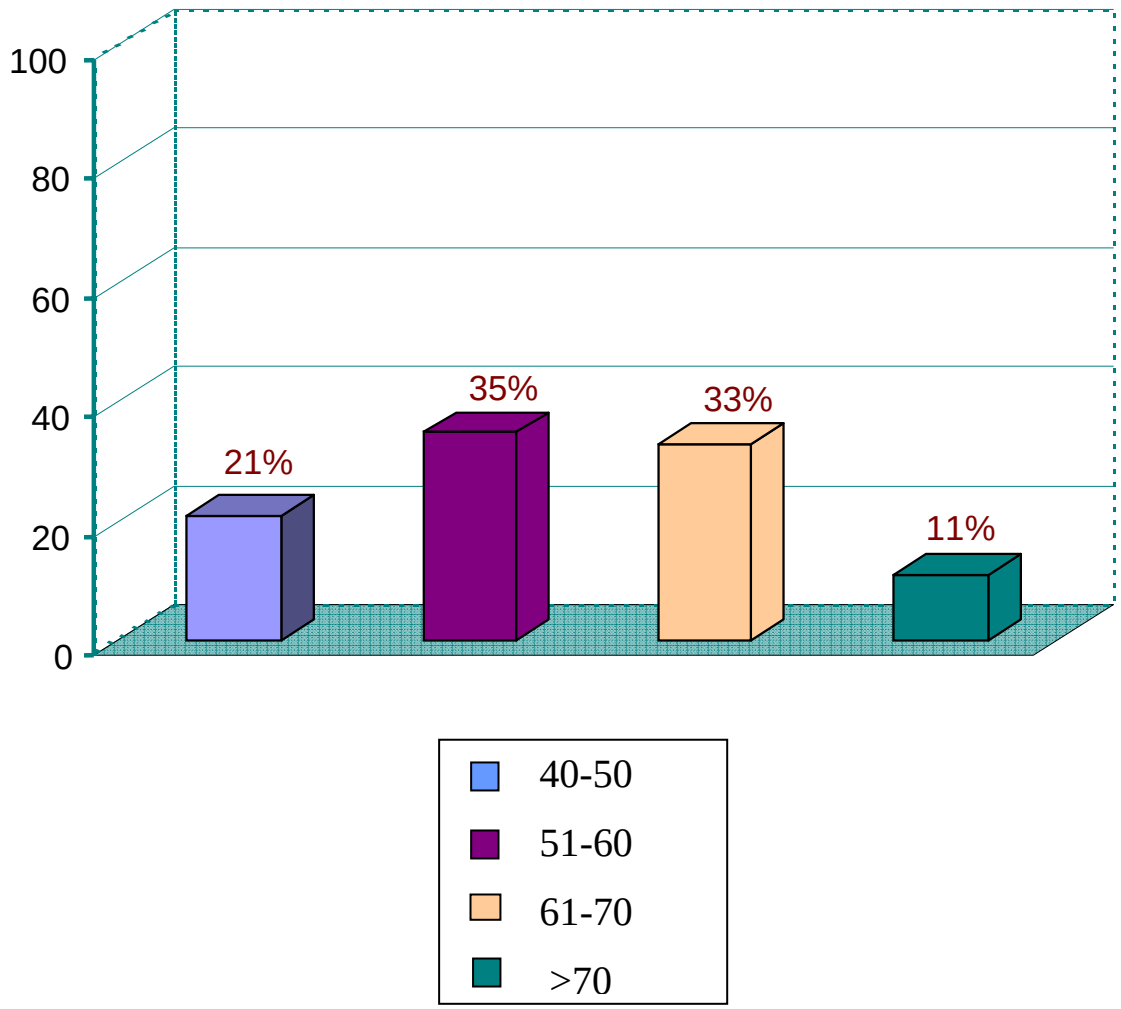

Figure (1):- Distribution of the 100 Sudanese female with MI according to their age.

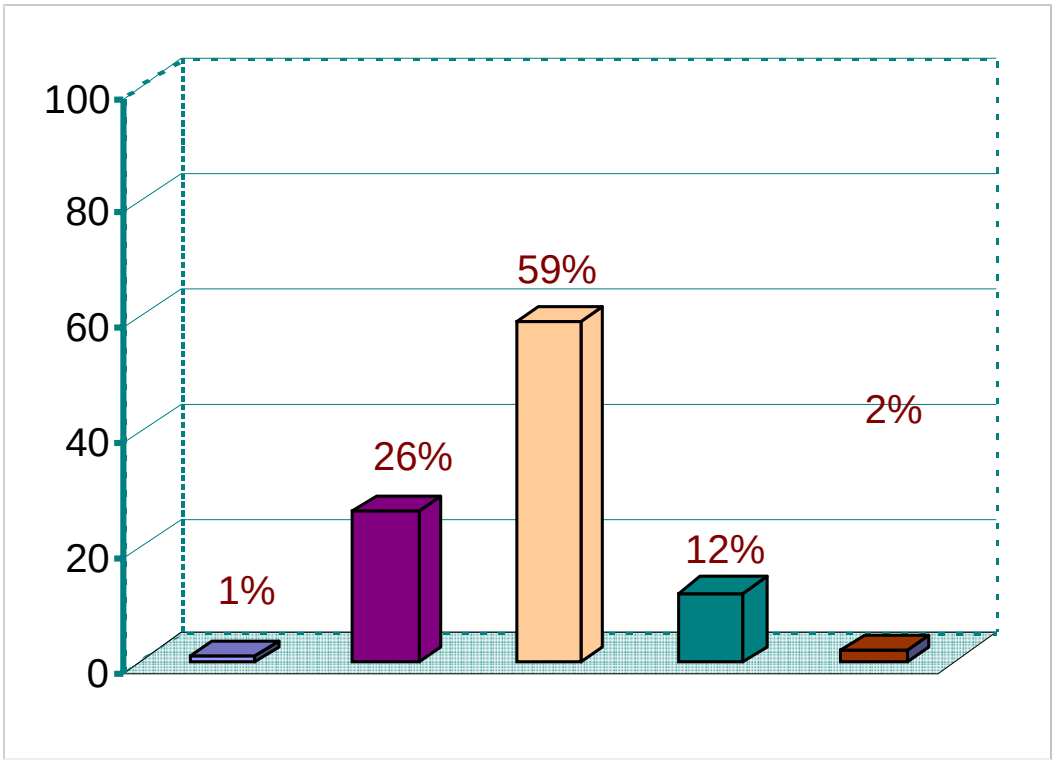

Figure (2):-Distribution of the 100 Sudanese female with MI according to their tribe.

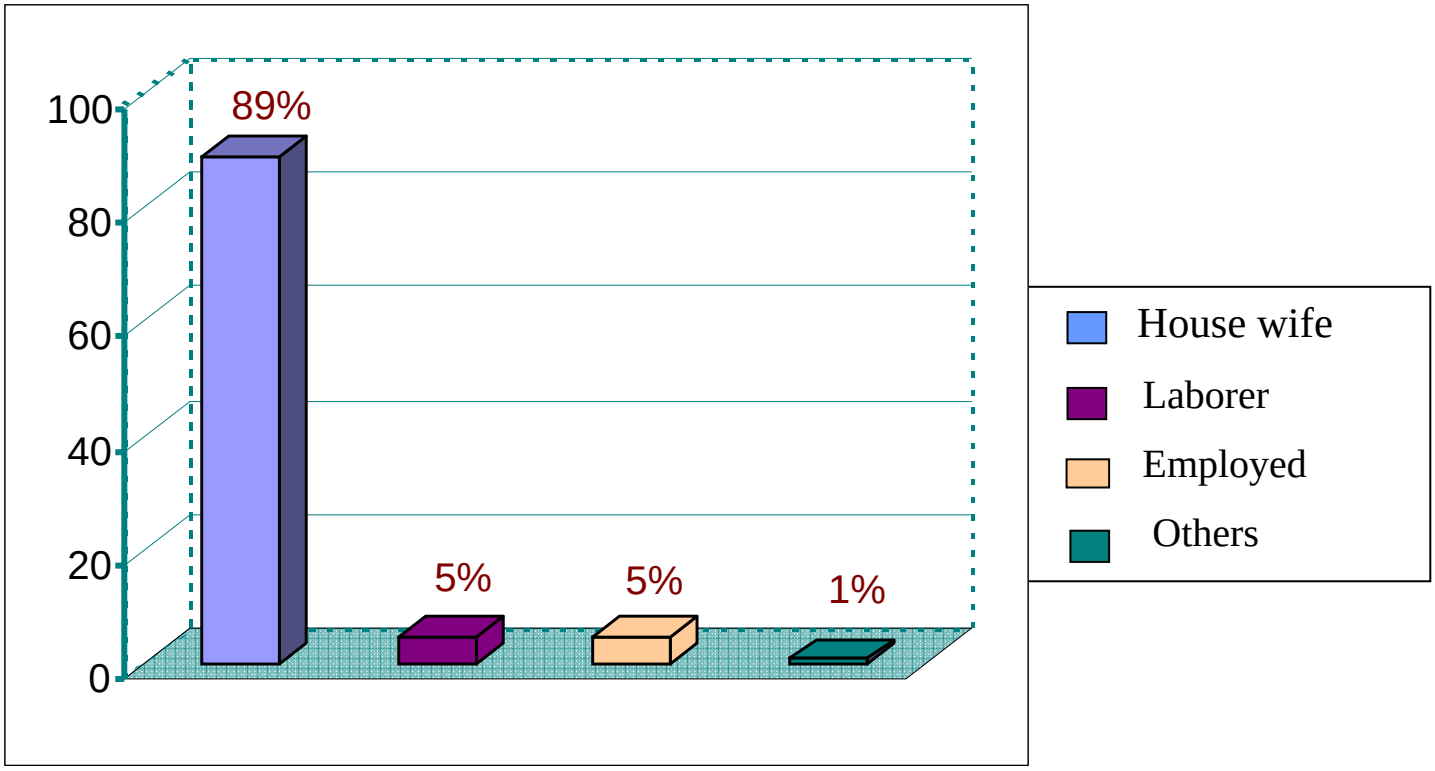

Fig (3) :- Distribution of a 100 Sudanese female with MI according to their occupation

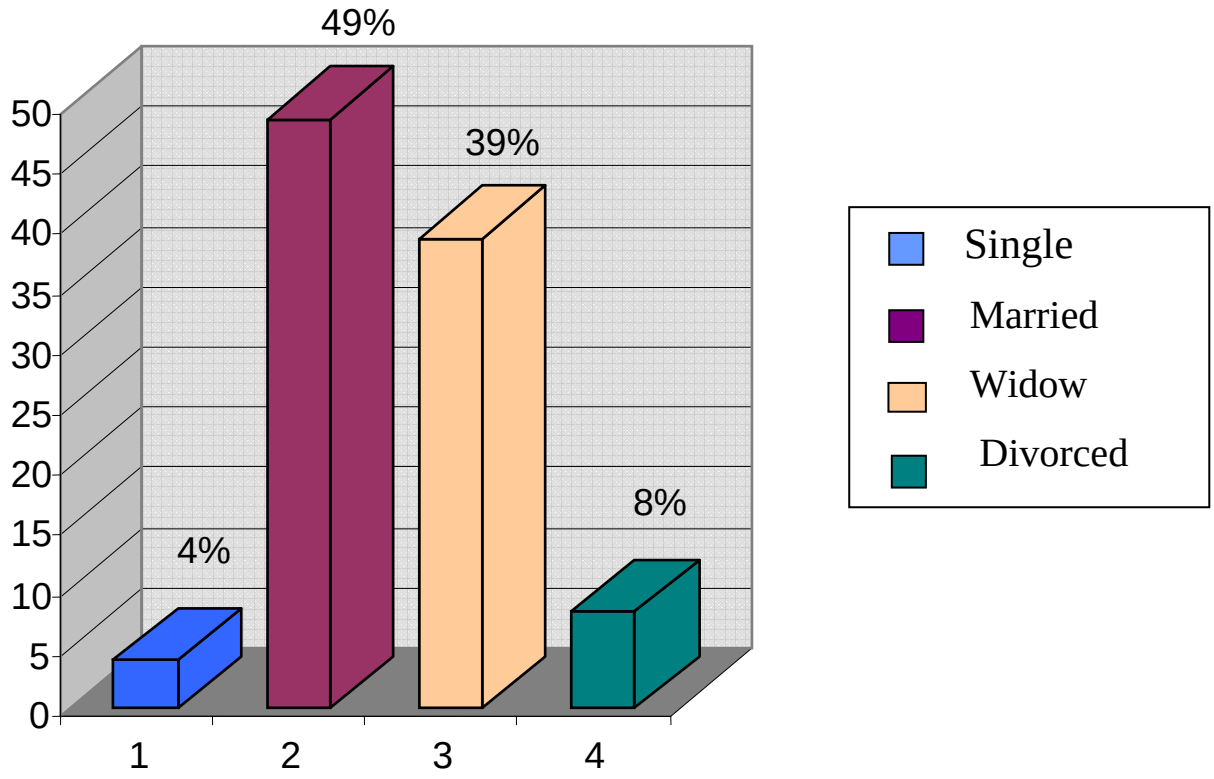

Fig (4) :- Distribution of a 100 Sudanese female with MI according to the marital status

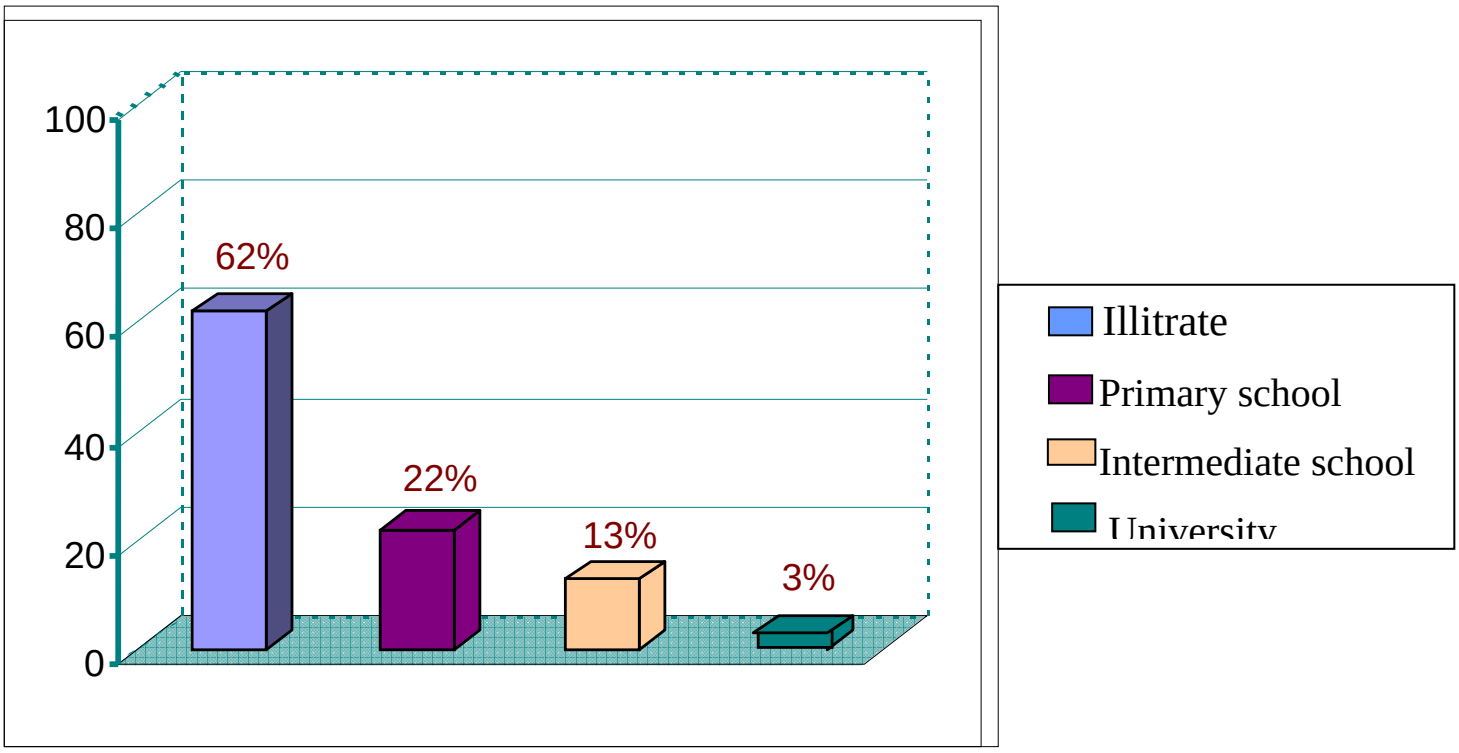

Fig (5) :- Distribution of a 100 Sudanese female with MI according to their education level

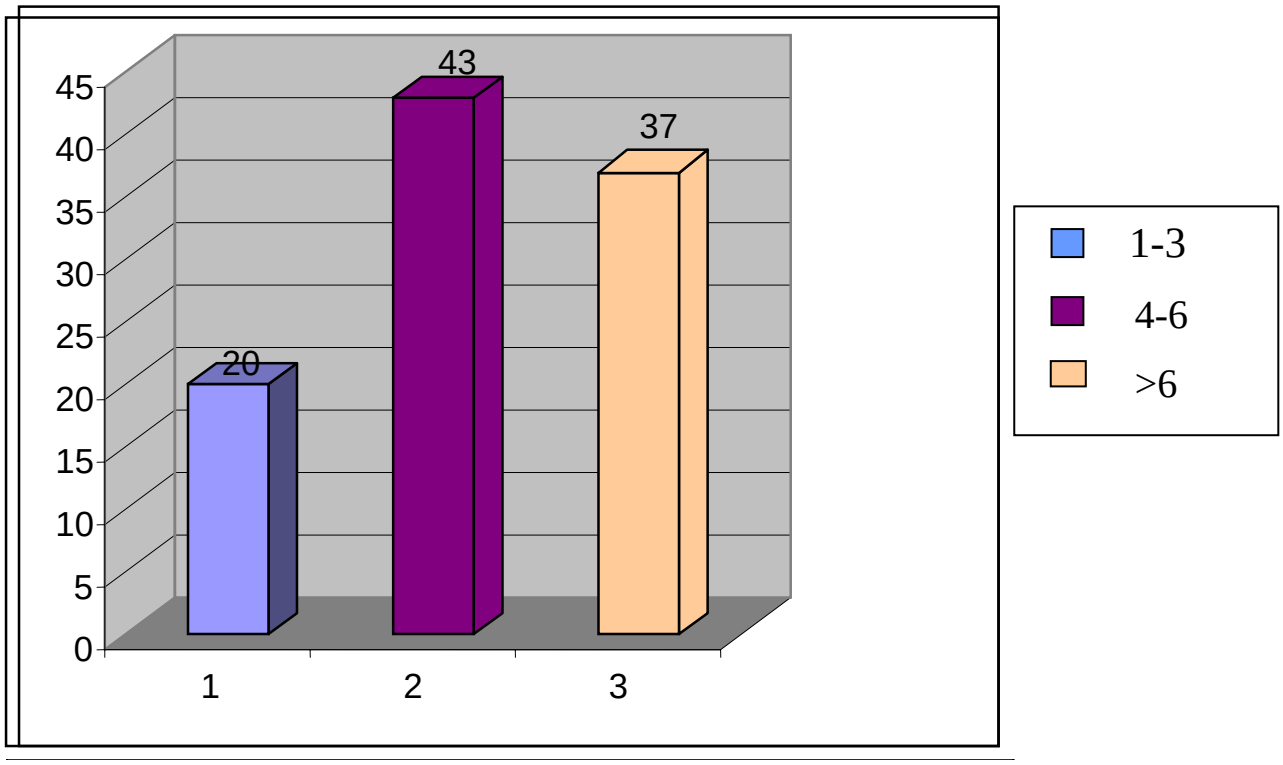

Fig (6) :- Distribution of a 100 Sudanese female with MI according to their number of pregnancy

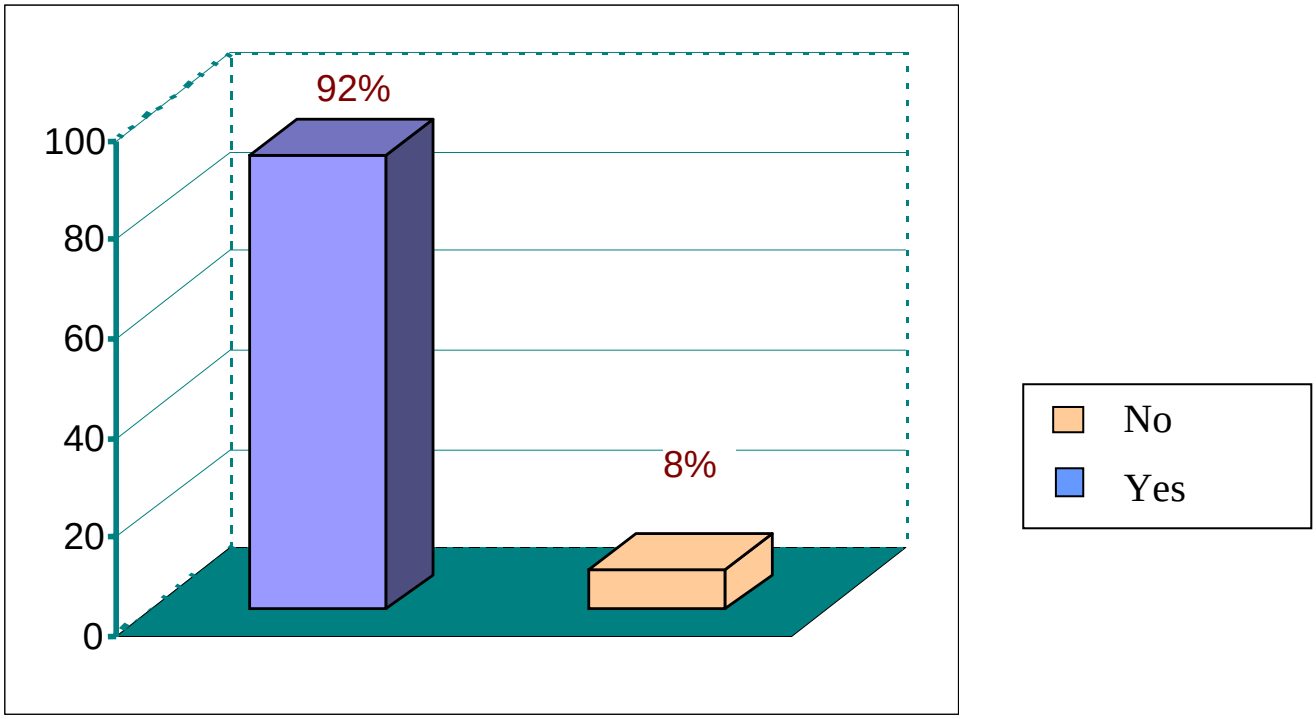

Fig (7) :- Distribution of a 100 Sudanese female with MI according to their chest pain

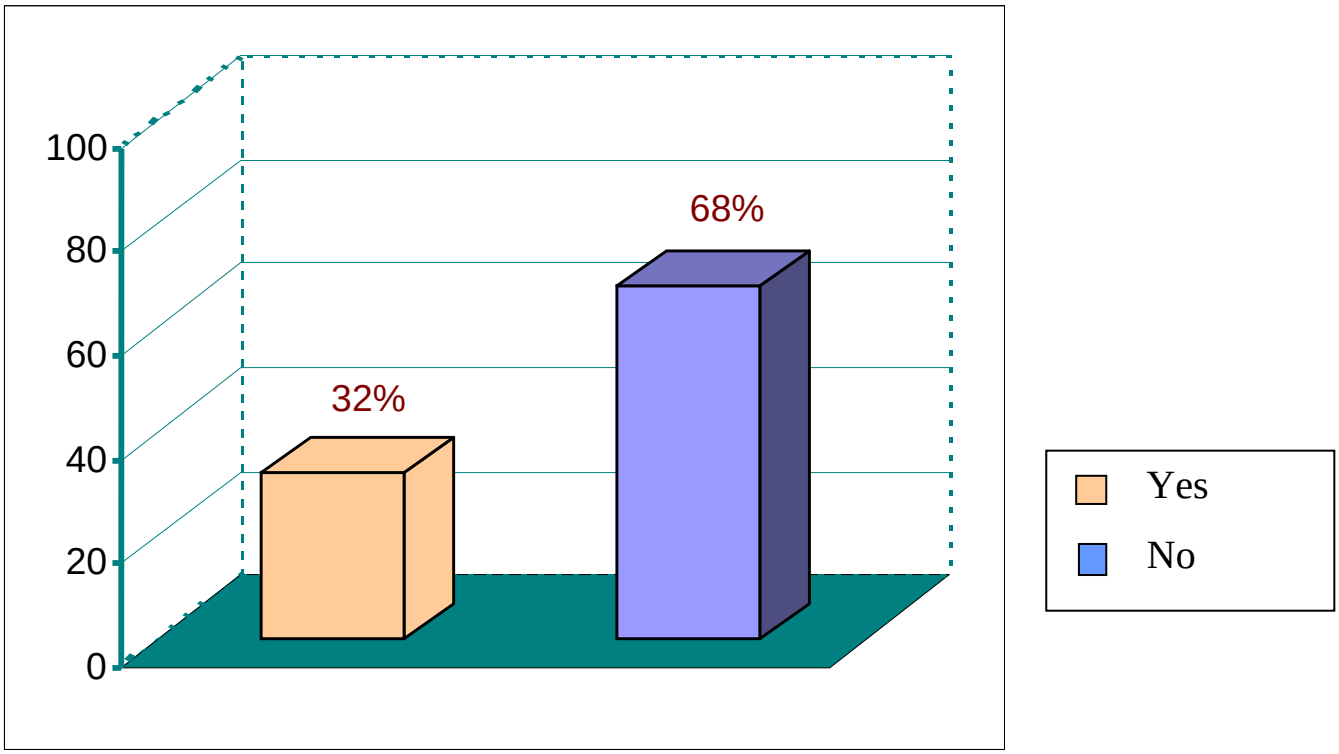

Fig (8) :- Distribution of a 100 Sudanese female with MI according to their shortness of breath

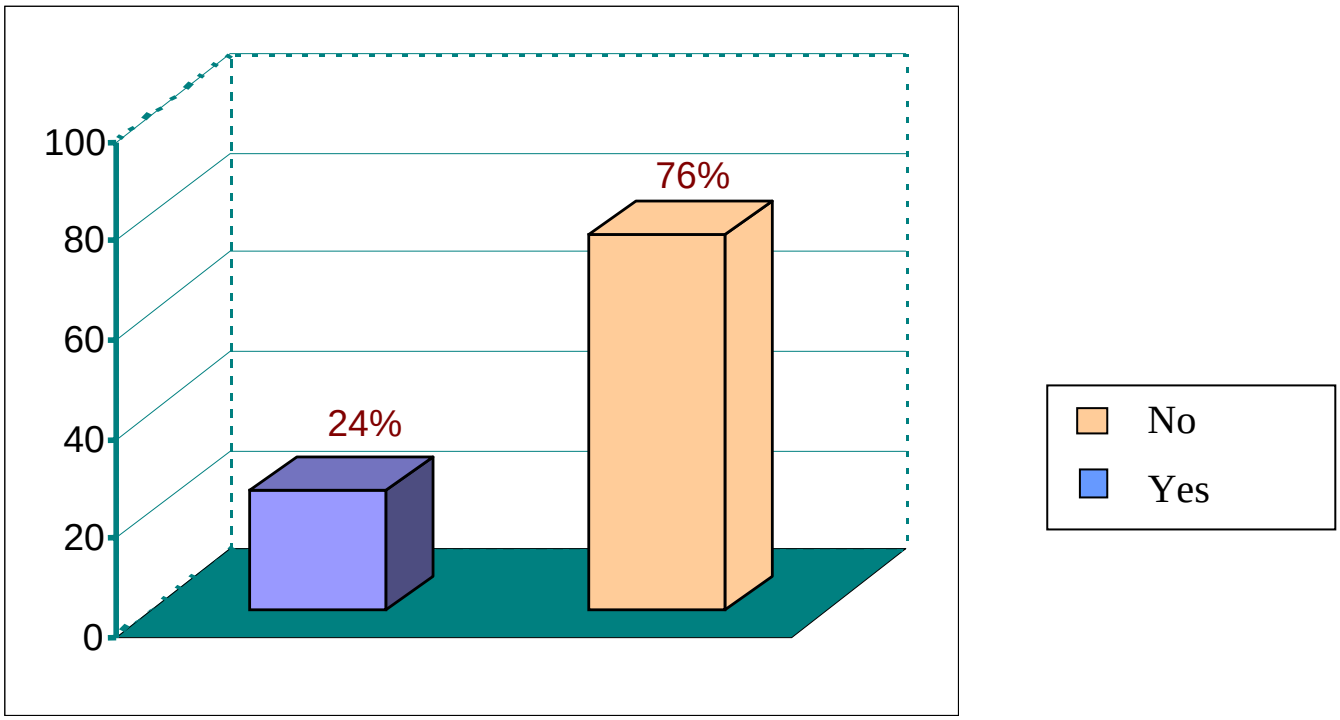

Fig (9) :- Distribution of a 100 Sudanese female with MI according to their palpitation

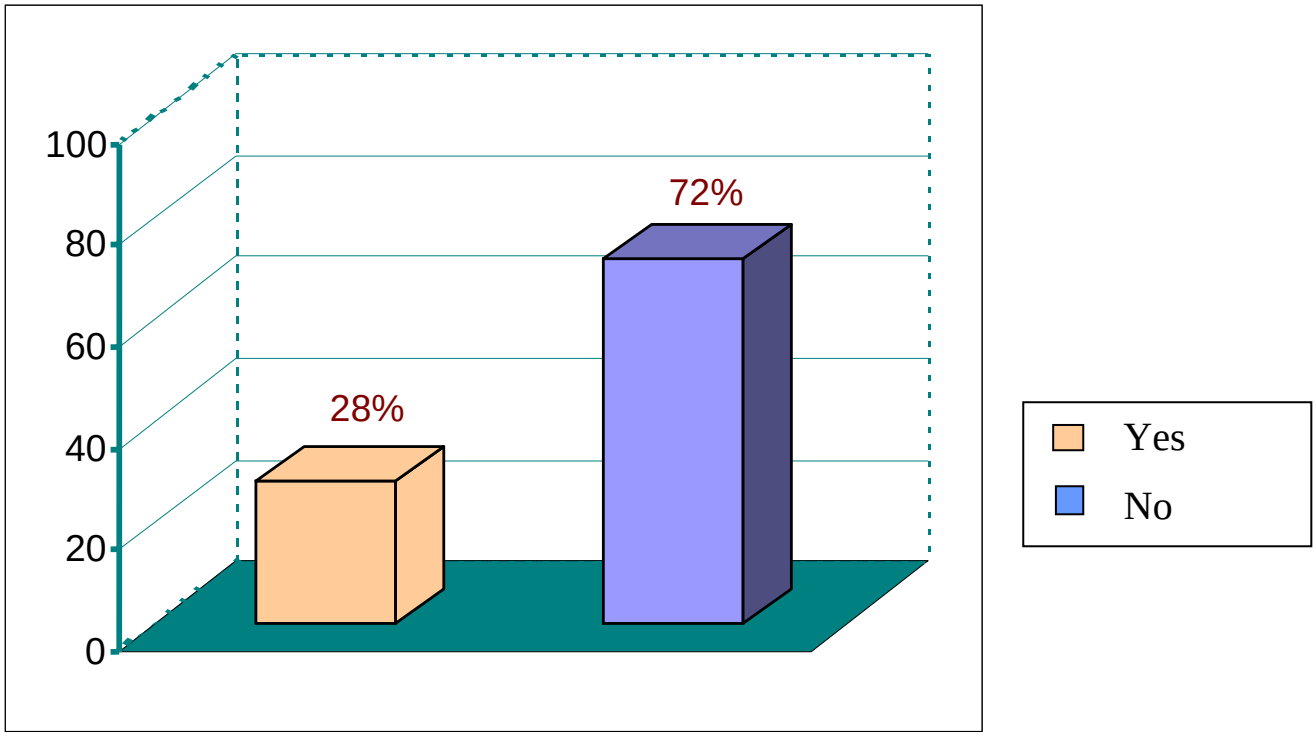

Fig (10) :- Distribution of a 100 Sudanese female with MI according to their family history

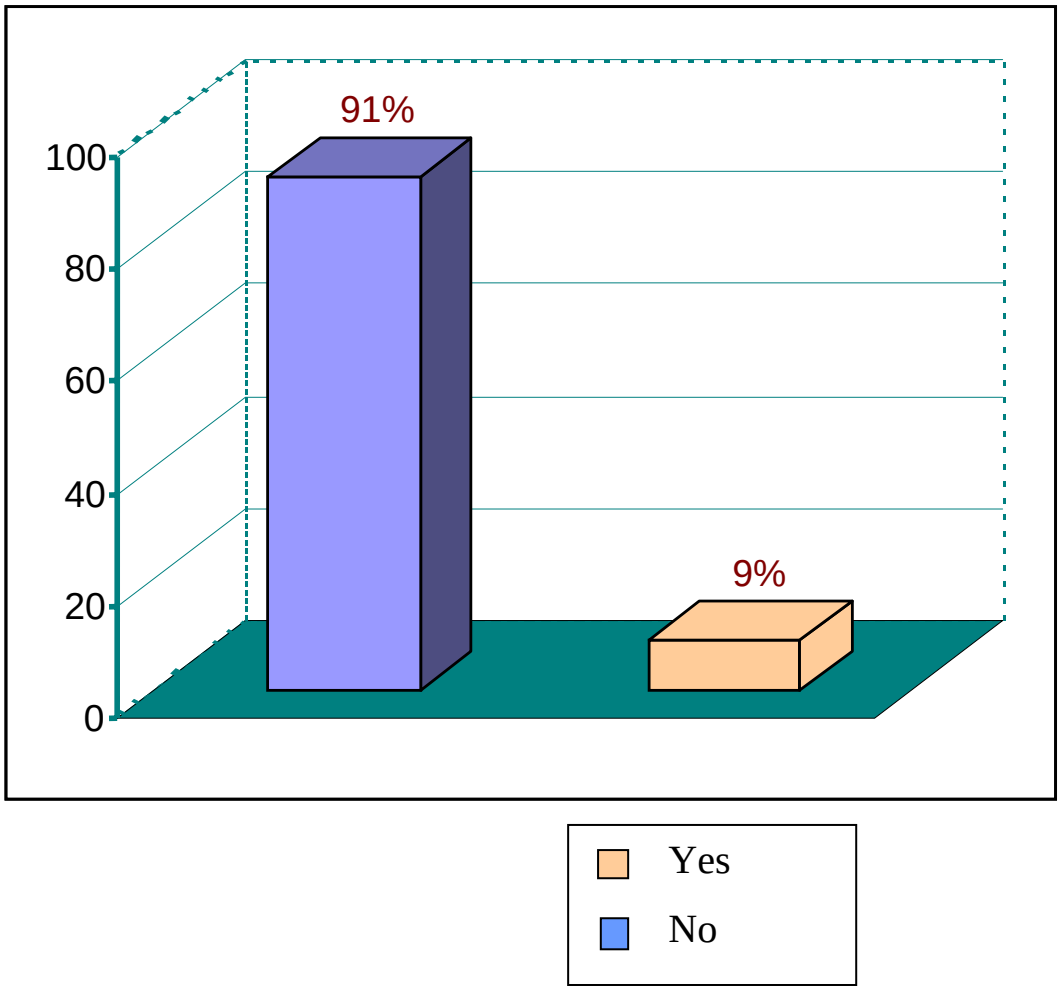

Fig (11) :- Distribution of a 100 Sudanese female with MI according to their cigarette smoking

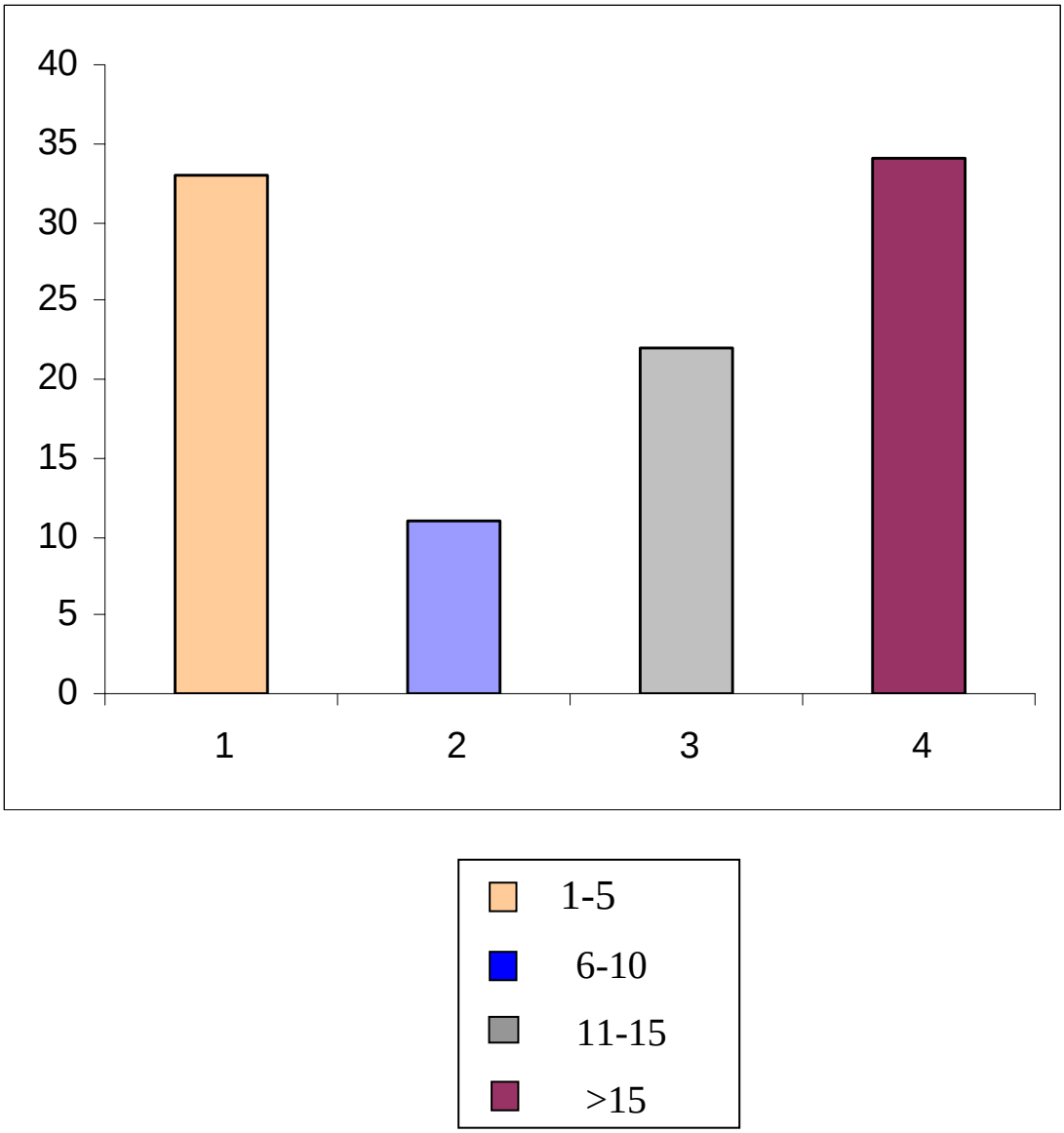

Figure (12):- Distribution of a 100 Sudanese female with MI According to the duration of smoking

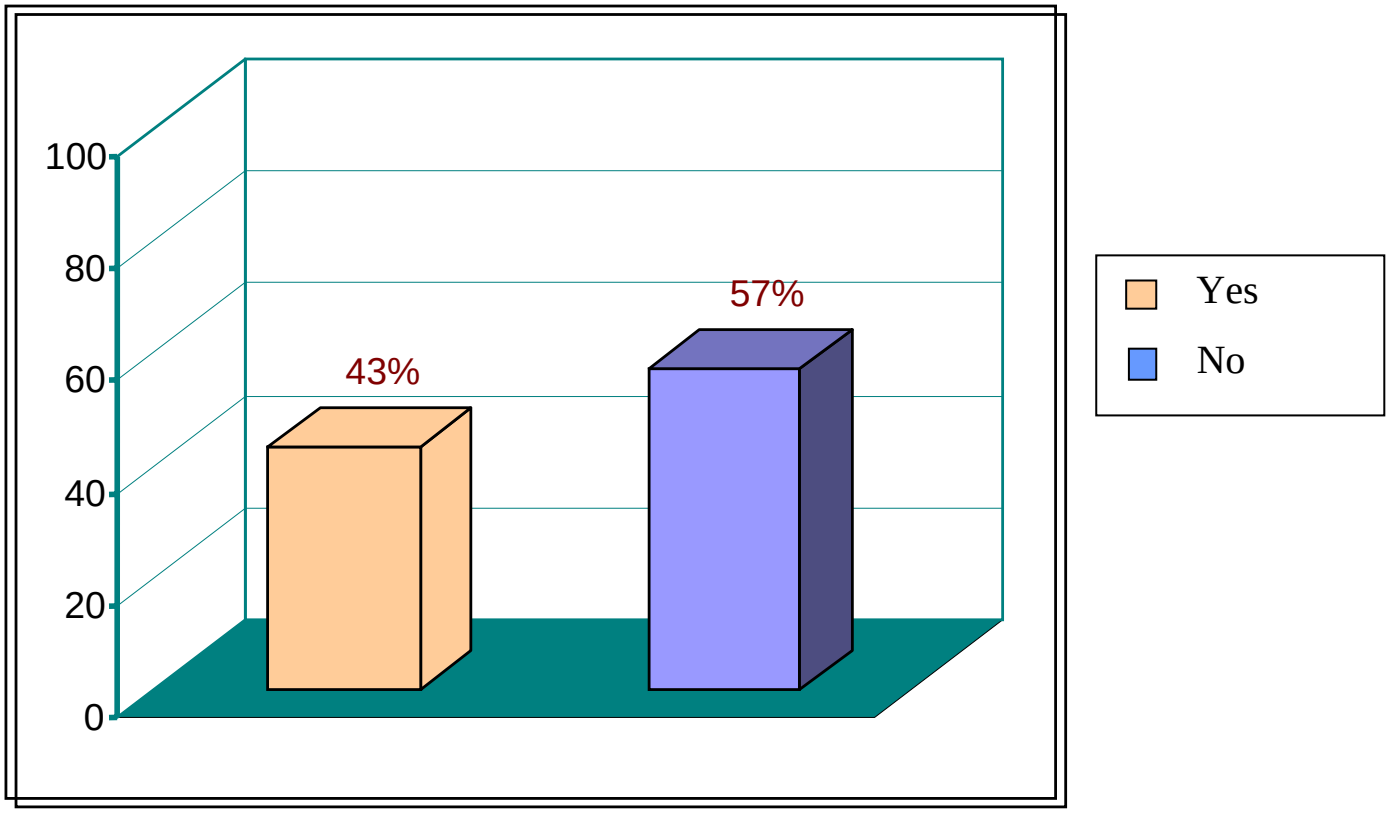

Fig (13) :- Distribution of a 100 Sudanese female with MI according to their systemic hypertension

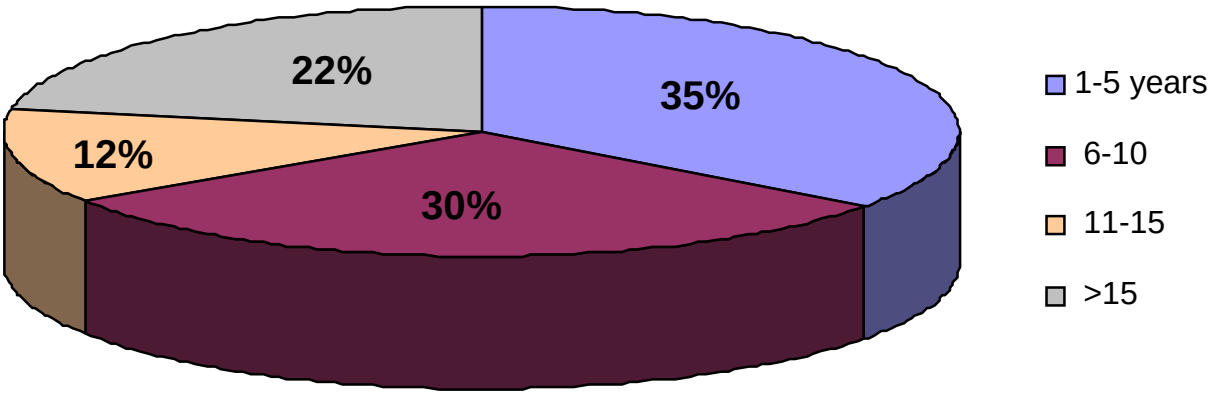

Fig (14) :- Distribution of a 100 Sudanese female with MI according to the duration of hypertension

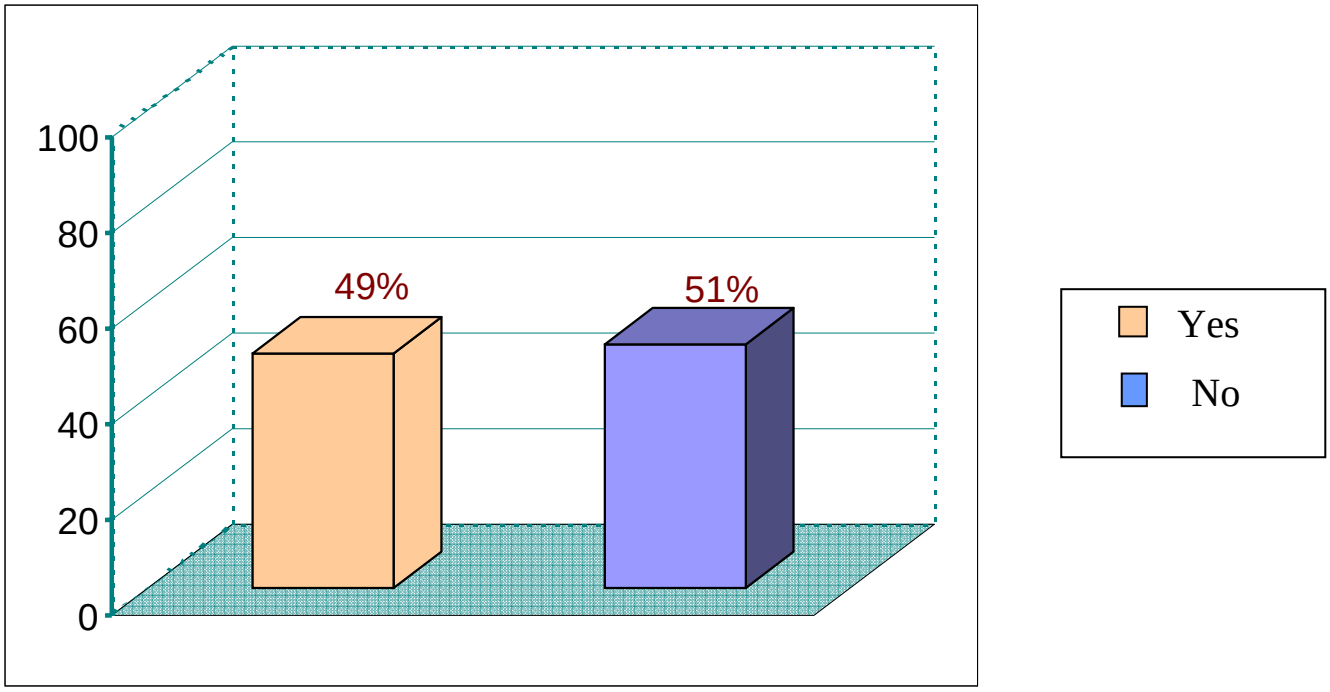

Fig (15) :- Distribution of a 100 Sudanese female with MI according to their Diabetes Mellitus

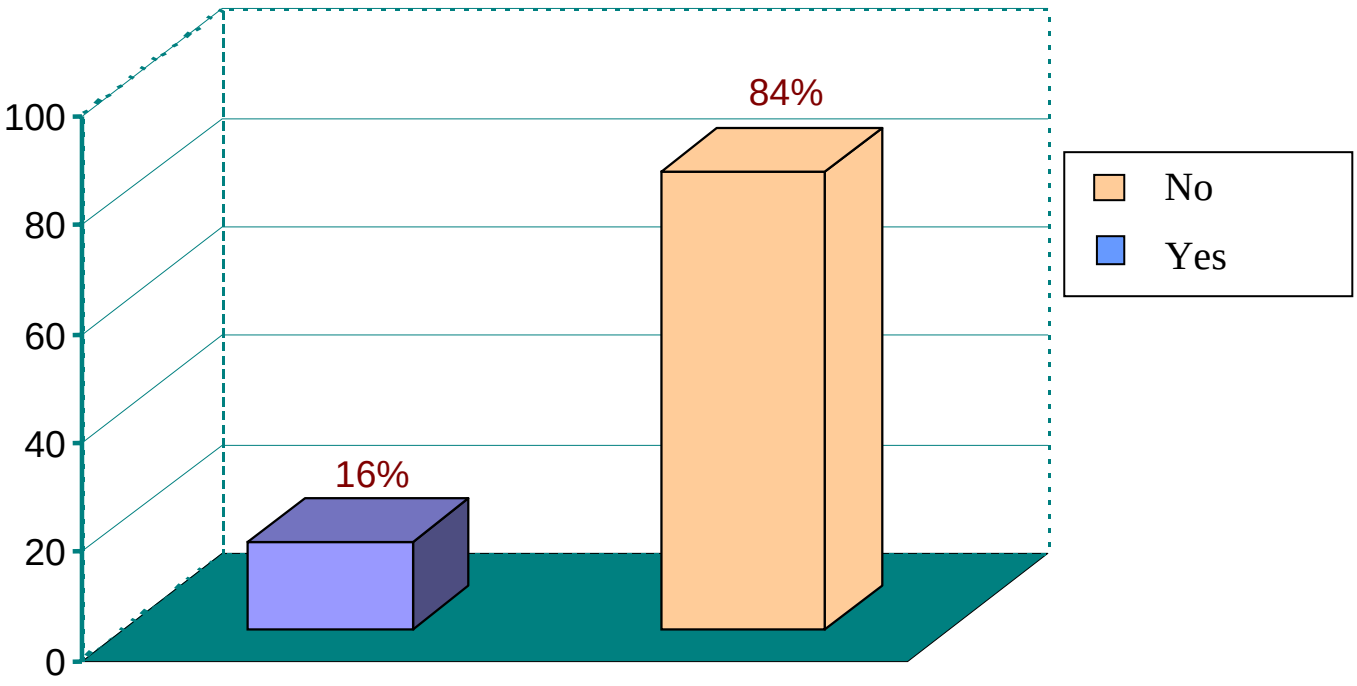

Fig (16) :- Distribution of a 100 Sudanese female with MI according to their pallor

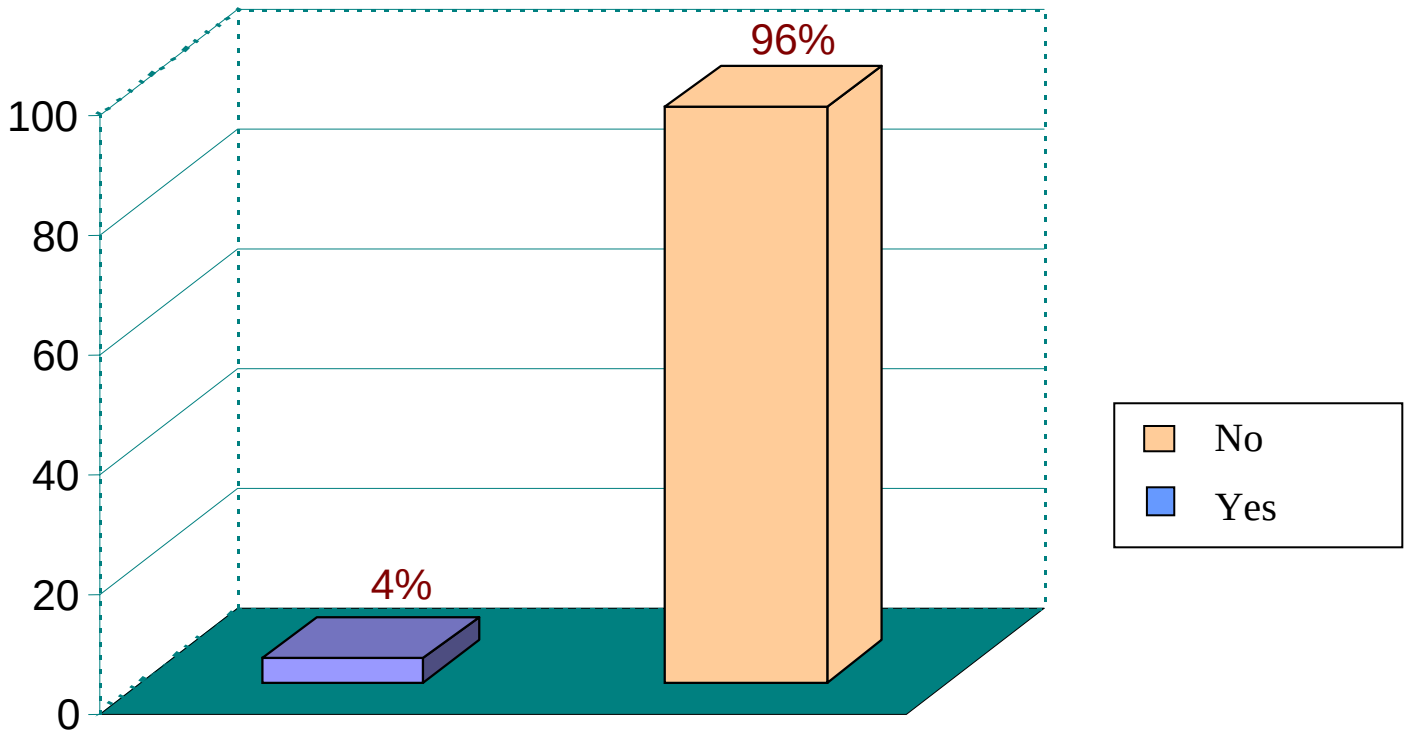

Fig (17) :- Distribution of a 100 Sudanese female with MI according to their signs of hyperlipidaemia

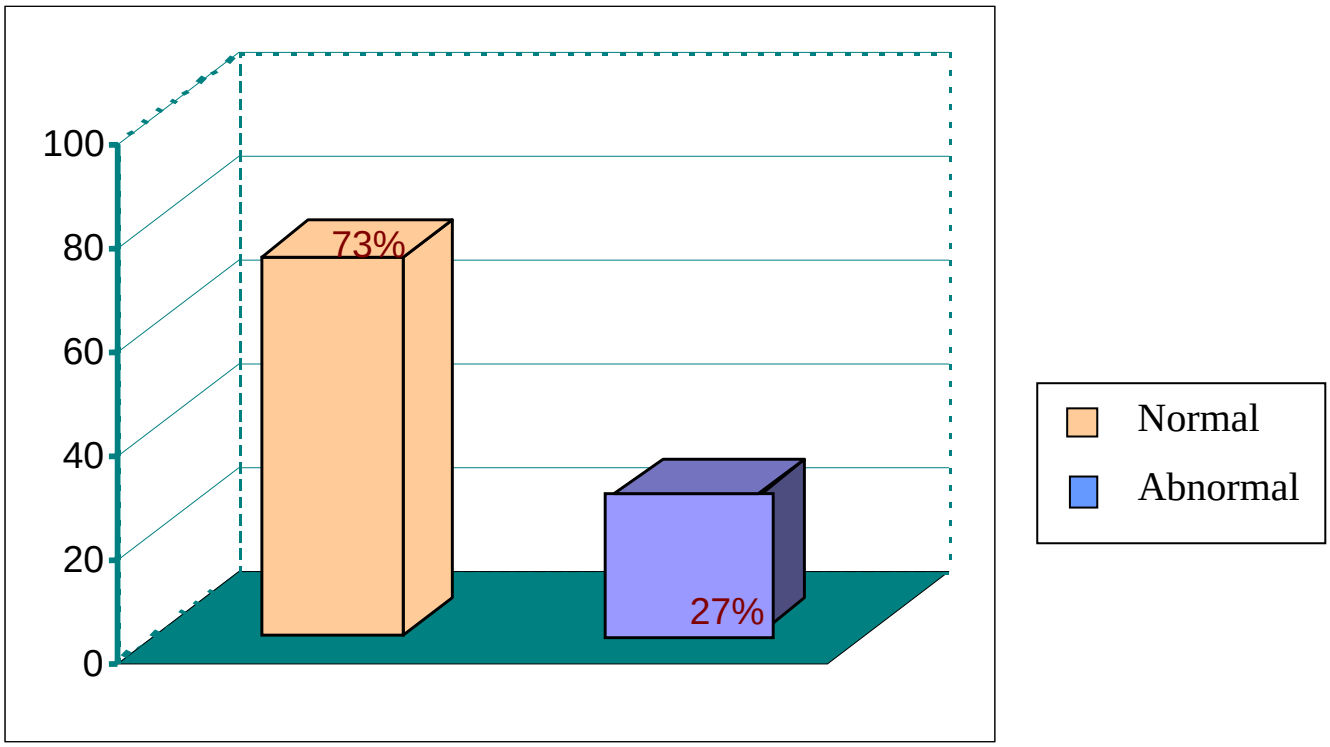

Fig (18) :- Distribution of a 100 Sudanese female with MI according to their lipid profile

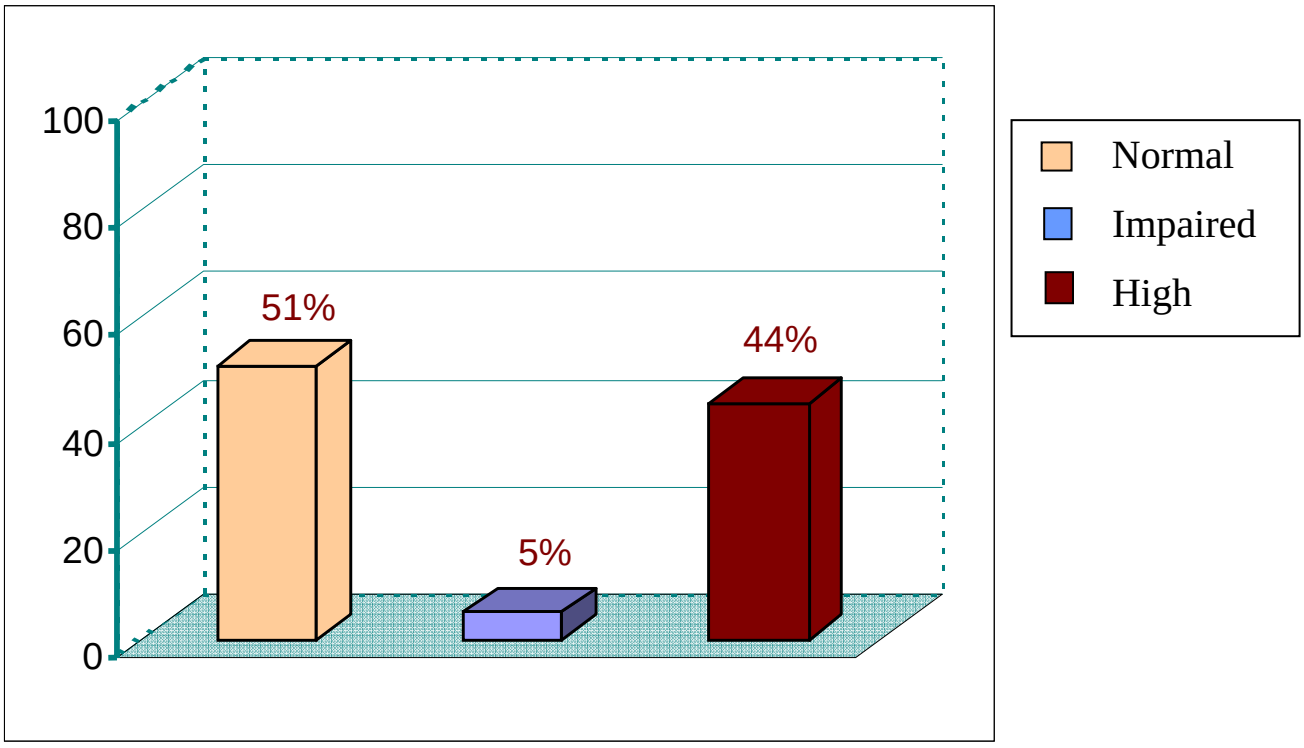

Fig (19) :- Distribution of a 100 Sudanese female with MI according to their RBG

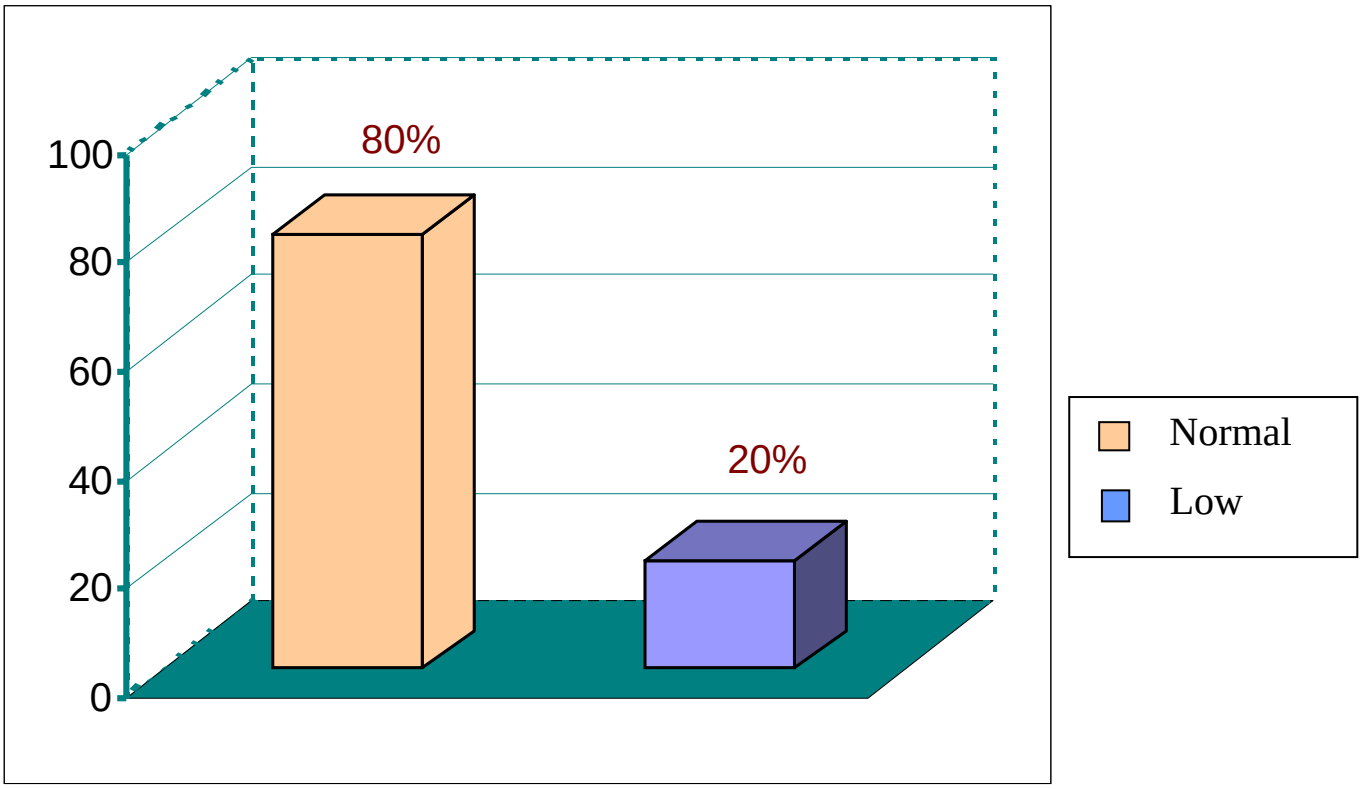

Fig (20) :- Distribution of a 100 Sudanese female with MI according to their HB

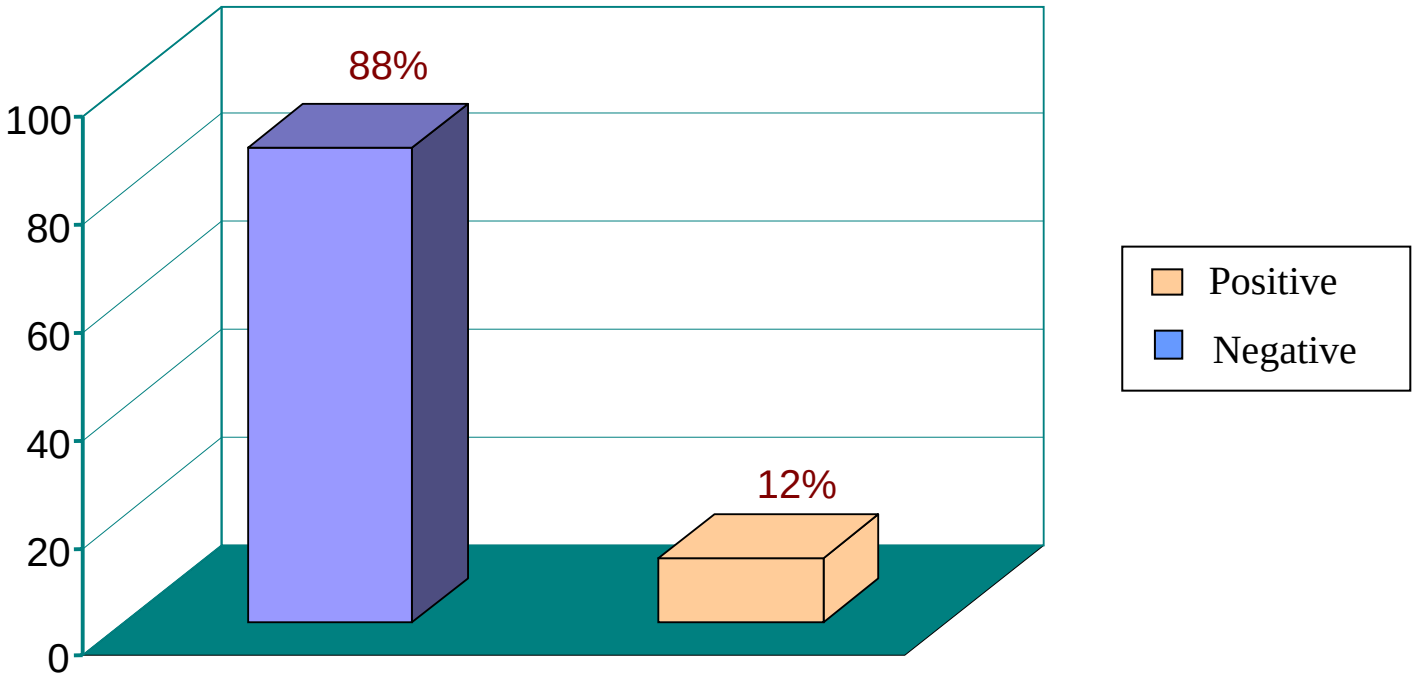

Fig (21) :- Distribution of a 100 Sudanese female with MI according to their BFFM+ICT for Malaria

Table (1)

Distribution of the 100 Sudanese female with MI according to their duration of Diabetes.

Diabetes duration	Percent
1-5years	15
6-10years	20
11-15years	30
>15years	35

Table (2)

The relation between the age and the presence of Diabetes in the studied group.

Distribution of age	Diabetes Mellitus
40-50	24%
51-60	33%
61-70	33%
>70	10%

Table (3)

The relation between the age and the smoking in the studied group.

DISTRIBUTION OF AGE	CIGARETTE SMOKING
40-50	11
51-60	44
61-70	33
>70	12

Table (4)

The relation between the age and the hypertension in the studied group.

DISTRIBUTION OF AGE	SYSTEMIC HYPERTENSION
40-50	19
51-60	35
61-70	35
>70	11

Table (5)

Distribution of the study population according to their BP level.

BP	PERCENT
Hypertension	34
Normal BP	60
Hypotension	6

Table (6)

Co-relation between pallor and the age of the 100 Sudanese female with MI.

Distribution of age	Pallor
40-50	12%
51-60	19%
61-70	50%
>70	19%

Table (7)

Distribution of a 100 Sudanese females with MI presented with Arrthymia.

Arrthymia	**Percent**
Yes	3%
No	97%

Table (8)

Distribution of a 100 Sudanese females with MI presented with signs of heart failure.

Signs of heart failure	Percent
Yes	6%
No	94%

Table (9)

The types of MI.

FEATURE Of ACUTE TRANSMURAL	PERCENT
ANTERIOR	55
POSTERIOR	7
INFERIOR	38

Table (10)

The level of cardiac enzymes.

Cardiac enzymes	Percent
Normal	42
Abnormal	58

DISCUSSION

Coronary heart disease (CHD) is a common disease and cause of death in women, accounting for over 250.000 deaths in women per year. Over the last two decades, multiple important studies have helped define accurate clinical tests, risk factors, preventive interventions and effective therapies for CHD.

Unfortunately, many of these studies have either excluded women entirely or included only limited numbers of women and minorities.

Thus, much of the evidence supporting contemporary recommendations for testing, prevention, and treatment of coronary disease in women is extrapolated from studies conducted predominantly in middle-aged men. The two best approaches to obtain additional evidence on diagnosis and treatment of CHD in women are to conduct large studies that include adequate numbers of women and minorities to answer the research question or to perform systematic reviews and meta-analyses summarizing effect estimates by sub group.

According to Figure 1, 21% of the cases in the study lie in the age group (40-50), 38% in the age group (51-60), 33% in the age group (61-

70) and 11% in the age group more than 70 years. In conclusion, approximately two third of the patients (68%) lie in the age group ranging from (51-70), and this is consistent with the fact that estrogenic as cardioprotective so the incidence of M.I increases markedly at menopause.

The majority of the patients were housewife (89%), while 5% employed and 5% labourers. (Figure 3)

Less than half (49%) of the patients were married, while 39% were widows followed by 8% divorced and 4% single. This result showed that chronic psychological distress may be associated with acute coronary syndromes. (Figure 4) As in a study done on 1998 company130 patients with an acute MI or unstable angina to 102 control subjects hospitalized for acute trauma found that cardiac patients had higher levels of social isolation, self-blame, avoidance, and more painful life experiences compared with controls. High levels of psychologic distress were detectable on about 75% of cardiac patients. [39]

The majority of the patients (62%) were illiterate, where 22% had a primary school education level, 13% intermediate school and only 3% complete the university. (Figure 5)

The number of pregnancies in the patients were 20% (1-3), while 43% (4-6) and 37% had more than 6. (Figure 6)

Chest pain in the presenting complains in 92% of patients, followed by shortness of breath in 32%, while 24% presented with palpitation. (Figure 7, 8, 9)

The family history only accounts for 28% in patients. (Figure 10)

The percentage of the patients who used to smoke were 9%, but the real percentage may be higher since this is forbidden socially. (Figure 11)

According to the results in figure (12), the duration of smoking is not related to MI in those who used to smoke. 77% of those who used to smoke lie in the age group (51-70) years. (Table 3)

The percentage of the patients who had systemic hypertension (HTN) was 40%. (Figure 13)

35% of them had HTN for (1-5) years, followed by 30% (6-10) years while 12% (11-15) years and 23% for more than 15 years. (Figure 14)

The majority of the patients (70%) lie in the age group (51-70) years, which was the main age group of the patients. (Table 4)

46% of the patients had DM, of whom 15% had the disease for (1-5) years, 0% (6-10) years, 30% (11-15) years and 35% for more than 15 years. This shows that the duration of DM is a risk factor for M.I. (Table 1 & Figure 15)

The percentage of the patients who had anemia on admission were 16%, 69% of them lie in the age group (51-70) years. (Figure 16 & Table 6)

The majority of the patients who had a normal BP (60%), while (34%) had a high BP and only (6%) were hypotensive. (Table 5)

Only 4% of the patients had signs of hyperlipidaemia on presentation. (Figure 17)

According to the investigations done the majority of the patients (73%) had a normal lipid profile. (Figure 18)

Most of the available data on cholesterol and M.I were derived from studies of middle age men in whom it had been observed that for every 1% decrease in total serum cholesterol, there is (2-3)% reduction in risk of M.I similarly the risk of M.I increases by (2-3)% for every 1 mg / dl decrease in HDL cholesterol, also low HDL cholesterol levels in women may be more predictive for subsequent coronary events than high levels of HDL cholesterol. [40]

80% of the patients had a normal HB % and only 12% had a positive BFFM. (Figure 20, 21)

At time of presentation 51% of the patients had a normal blood glucose level, 44% had high blood glucose and 5% had impaired test. (Figure 19)

There is a positive association between hyperglycemia at the time of an M.I and mortality. The evidence that the use of insulin to lower blood glucose concentration reduces the mortality in patients with diabetes suggests that hyperglycemia is not only an epiphenomenon of the stress response mediated by sympathetic stimulation. [41]

3%of the patients presented with arrthymia and 6% with signs of heart failure. (Table 7 & 8)

55% of the patients had an anterior M.I while 38% had an inferior M.I and only 7% had posterior M.I. (Table 9)

58% of the patients had abnormal results of cardiac enzymes at time of presentation (CK-MB). (Table 10)

The prognostic significance of the small elevation of (CK-MB), which represents an evidence of myocardial damage, in patients with unstable Angina is uncertain. The relationship between peak (CK-MB) level and outcome in patients with an acute coronary syndrome unassociated with ST segment elevation was evaluated in the PURSUIT trial of 8250 patients from whom at least one (CK- MB) reading was obtained. The mortality was related to the presence of an elevated (CK-MB) level and the degree of evaluation. [41]

CONCLUSION

In conclusion, it was found that large number of the patients present after the age of 50 years.

The majority of patients were multipara (more than 6 pregnancies).

The commonest presenting complain was chest pain.

To be more precise the diabetes mellitus was the main risk factor.

Quite small number of patients were found to be anaemic (only 16 %).

More than half of the patients had elevated cardiac enzymes (CK-MB).

The majority of the patients present with Anterior MI, then Inferior MI and Posterior MI.

Recommendations

- All female patients should undergo aggressive control for their diabetes and hypertension.
- All patients should undergo aggressive reduction for dyslipidemia and even patients with high-normal serum cholesterol concentrations have been shown to benefit from lipid lowering.
- All female should encourage stopping smoking.
- Early assessment and management of the high risk patients.
- Further long term prospective studies on larger number of patients are warranted to evaluate the risk factors and to assess its role in predicting future MI in females.

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RESULTS

This study was conducted on 100 female patients with myocardial infarction. 21% of the patient between 40-50 years, 35% between 51-60 years, 33% between 61-70 years and 11% more than 70% years **(Figure 1).**

Most of the patients 59% originally from Northern Sudan, 26% from Central, 21% from Western, 2% from Southern and 1% from Khartoum **(Figure 2)**.

89% of the patient were housewife, while 5% employed, 55% labourer and 1% others **(Figure3).**

The patient were married is 49%, widow 39%, divorced 8% and 4% were single **(Figure 4)**.

The patient were illiterate 62%, 22% had a primary school education level, 13%, intermediate school and only 3% completed the university studies **(Figure5).**

The distribution of the patients according to their number of pregnancies were 20% 1-3, 43% 4-6 and 37% > 6 **(Figure6)**.

Distribution of presenting symptoms, 92% presented with chest pain, 32% presented with shortness of breath and 24% presented with palpitation **(Figure 7, 8, 9).**

The patients that had a family history of ischemic heart disease is only 28% **(Figure 10)**.

The risk factors of smoking are shown in **(Figure 11, 12**), 9% of the patients used to smoke and duration more than 10 years in 55% of patients.

(Table 3) showed that who used to smoke were 11% in the age group 40-50, 44% in 51-60 years and 12% more than 70% years.

The distribution of systemic hypertension as a risk factor is shown in **(Figure 13-14)**, 40% had systemic hypertension and duration more than 15 years in 23%.

(Table 4) showed that those were known to be hypertensive were 19% in age group 40-50 years, 35% in 61-70 years and 11% more than 70 years.

Diabetes mellitus occurred in 46% with a duration of more than 15 years in 35% **(figure 15 and table 1)**, 10% of them are in age group more than 70% years **(table 2)**.

Distribution of presenting signs, 34% presented with high blood pressure, 4% had a sign of hyperlipdaemia and 16% of the patient were pale on admission **(Figure 16-17)**.

(Table 6) showed that 12% of the patients in the age group 40—50 presented with pallor, 19% in 51-60 years, 50% in 60-70 years and 19% more than 70 years.

Only 3% of the patient presented with arrthymias and 6% presented with sign of heart failure **(table 7 and 8**).

Random blood sugar was high in 44% **(figure 19).** Normal Hb% in 80% and 20% had low Hb% **(figure 20).** BFFM positive in 12% of the patients while 88% had negative result **(Figure 21%).**

ECG showed 55% of the patients had anterior MI, 38% had inferior M.I, while 7% had posterior M.I **(table 9).**

Cardiac enzyme are high in 42%, while 58% had abnormal result **(table 10)**.